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PHYSICAL DIAGNOSIS *of* DISEASES *of the* CHEST

By

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and

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WITH 166 ILLUSTRATIONS

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

1925

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1925

PREFACE

This treatise has been written in the hope of utilizing the experience gained in the instruction of medical officers in physical diagnosis at the Medical Reserve Officers' Camps during the late war. It was found in this work that many physicians who have paid no especial attention to physical diagnosis, if given proper instruction, become interested in the subject and develop a previously unsuspected facility.

It is unfortunate that in the teaching of physical diagnosis demonstrations are often made only on the sick, so that many pupils are never led to inquire whether physical signs noted in the sick and commonly believed to be pathological may not occur in the normal, as well as in the diseased body. The study of physical diagnosis should begin with the normal chest. For this reason the localized peculiarities of the chest in health have been discussed in detail in a special chapter, the treatment of the subject being somewhat full because many of the facts stated have not become familiar to the medical profession. The auditory phenomena of the costopleural sinus, for example, were demonstrated to many hundreds of pupils in the Army Schools who, almost without exception, heard of them for the first time.

In acquiring the art of physical diagnosis nothing of course can be a satisfactory substitute for practical instruction in small classes. Such success as was attained in the Army work was largely due to the fact that personal instruction was given by specially selected experts who supervised and directed the work of each student until he had mastered the first principles of auscultation and percussion, or had shown himself incapable of so doing.

In the absence of an instructor the student is urged to work with healthy subjects (small groups of students can examine one another to great advantage) until the characteristics of the breathing and of the percussion-note of the healthy chest, including the local variations, are thoroughly mastered before attempting to examine the subjects of pulmonary disease.

Usually in books devoted to the subject the laws of physical diagnosis are stated summarily and the student is expected to apply them, in fact, to work out his own methods. Experience shows that many who are intellectually capable of this feat do not do so. In view of the great

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demands which the study of medicine makes to-day upon the time and strength of the student it seems desirable that the methods of applying physical laws to diagnosis should be set forth, as fully as is practicable in a written treatise. For this reason the physical signs which characterize pulmonary lesions are described and discussed in some detail. This method which, the expert may think, leads to unnecessary restatement of laws and facts is believed to be of advantage to the beginner. It is the opinion of the writers that physical diagnosis should be, as far as practicable, standardized with a view to making it easy and thus, it is hoped, lead the medical practitioner to occupy himself seriously with the subject.

No attempt has been made to include obscure, rare or debated points of diagnosis. The aim has been, rather, to seek to induce the student to familiarize himself with the fundamentals of the art of physical diagnosis. After he has thoroughly learned percussion and auscultation he will be in a position to estimate the value of the many special methods which have been brought before the medical profession, whereas a premature introduction to the "refinements" of diagnosis would throw undue emphasis upon the new, the doubtful and the untested and would, therefore, tend to confuse and unsettle him.

In studying this book it is recommended that the student begin with the standard subjects, inspection, palpation, percussion and auscultation, making use of the chapters on the laws of sound and their application to the thorax as collateral reading and, after the first principles have been mastered and applied to the healthy chest under the guidance of the section which deals with its examination, returning to Chapter III for a more thorough study, after which the diagnosis of the individual diseases of the thoracic organs should be taken up.

No apology is made for the fullness of the treatment of the subject of pulmonary tuberculosis. This is believed to be justified, not only by the complexity and importance of the disease, but also because those who are its victims furnish an inexhaustible material for the study of physical diagnosis. After one has mastered the diagnosis of pulmonary tuberculosis in its various forms the diagnosis of other diseases of the lungs is found to be relatively easy. On the other hand, one who has not reached such proficiency can hardly be said to be competent to practise internal medicine.

Pulmonary tuberculosis can neither be diagnosticated nor treated with success unless the physician is able to visualize the pathology of the disease. For this reason and also in order that the standpoint of the

writer might be understood, reference has been made, as briefly as clarity would permit, to the pathological conditions that underlie physical signs.

A considerable amount of new material has been made use of in the preparation of this treatise. Among the subjects discussed which are new or which have not as yet become familiarly known to the medical profession may be named the theory of the resonator action of the thorax based upon new physiological experimentation, the adventitious sounds of the costopleural sinus, of the chondrosternal articulations and of the sternum and the rôle of tuberculous lymphangitis in tuberculosis of the lung. The treatment of the subjects of the coin-test in pneumothorax and of the displacement of the mediastinum in unilateral disease of the lungs is original with the writer. New material has naturally demanded more explanation than subjects already well known, the result being that the fullness of treatment of a given subject from the pathological or physiological standpoint is such as is required sometimes by its relative unfamiliarity, rather than by its relative importance.

Reference to authorities has, in general, only been made when the statements have seemed to need support because of their newness or unusual character. The writer desires to express his special indebtedness in the preparation of the chapter on the transmission of sound through the chest of the works of Gerhardt, R. Geigel, F. v. Müller and Paul Martini.

C. F. Hoover is the authority on the significance in diagnosis of the movements of the costal arch in respiration. The chapter on this subject is based upon his articles in the second volume of Oxford Medicine.

In the sections on the examination of the heart a plan similar to that employed in the first part of the book has been followed. The study of the heart in health and disease is based in part on anatomy, but chiefly on normal and pathological physiology. It seems to the writer that knowledge of the functions of the heart is a prerequisite to the intelligent study of physical diagnosis of disease of this organ. For this reason the fundamentals of the newer physiology of the heart and circulation have been included and their bearing on the explanation of physical signs.

Throughout the work, however, the point of view in dealing with the subject is that of the clinician whose chief interest is the recognition of disease as it presents itself at the bedside and in the consulting room. Emphasis is placed on the simple methods of diagnosis that are available to every student and practitioner—inspection, palpation, percussion and

auscultation—and to them much space has been devoted. These methods of examination are described in sufficient detail to enable the reader to utilize them fully in the study of his cases. Students and even practitioners are apt to forget that the seeing eye, the trained finger and the educated ear are still worth more to the examiner than all the instrumental means of diagnosis.

The advance of science has enriched clinical diagnosis by the discovery of new methods of studying the heart and circulation, but these are to be used as accessories and not as substitutes for the simpler physical methods. Chapters are devoted to the radiographic examination, to blood-pressure determinations and to various graphic methods. No attempt should be made to minimize the importance of these newer methods. Their use has brought great additions to knowledge of the function of the heart. They made it possible to gain information from palpation of the pulse and from percussion and auscultation of the heart that was hidden from earlier generations of physicians.

The object of the physical examination should be something more than the mere diagnosis of cases. It is hoped that this work will stimulate the student in the wider study of disease, and teach him to interpret physical signs in their relation to the functions of the heart.

The writer is indebted to Dr. Paul D. White for the excellent series of electrocardiograms and for valuable suggestions. Aid is also gratefully acknowledged to Dr. C. N. B. Camac for original illustrations from his collection, and to Dr. Alfred E. Cohn, Dr. John W. Dewis, Dr. Samuel A. Levine, Dr. Francis W. Peabody and Dr. Alfred C. Redfield for advice and criticism.

May, 1925

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PHYSICAL DIAGNOSIS OF DISEASES OF THE CHEST

THE LUNGS

CHAPTER I

ANAMNESIS AND CASE HISTORIES

In obtaining the patient's account of himself we inquire into his past history for such light as may be thrown upon the duration, or the mode of origin, or the causative factors of the disease that is known, or suspected, to be present. We should also find out something of the stock from which the patient sprang, and—perhaps most important of all—we should learn the physical and psychical reaction of the patient to disease. What is to be aimed at is the production of an objective medical history, uncolored by the subjective bias of the physician in favor of one or another group of diseases. The foregoing remarks apply, of course, with especial force to obscure cases of more or less chronic disease. But a record which should fulfil the conditions mentioned would be of inestimable benefit in the future to the family physician, though the condition for which treatment is demanded at the time be comparatively trivial. Conditions which seem trivial may, none the less, be the beginnings of serious disease.

The past history of the patient is not a dry catalogue of affections that befell him by chance. The nature, duration, and frequency of past attacks of disease give valuable information as to the sort of human being that one has to treat, the direction from which trouble is to be apprehended, what he is likely to die of, his emotional reaction to disease, and the like. Viewed in this way, the taking of a history becomes a fascinating psychological problem, and at the same time demands a breadth of view and an objective method of approach which rarely come instinctively to the physician. In fact, the art is one to be learned only by practice and close attention. The recorder should set about his task in an analytical spirit, seizing the important points, but at the same time not neglecting seemingly unimportant clues. Often the patient will let drop by chance a statement that throws a flood of light upon a difficult problem. The recording of the minutiæ, as given by the patient, is a

tedious and unprofitable task. The more voluminous the records, the less likely is it that the physician will continue, in the stress of practice, to make them. If the salient points are unerringly seized upon, the history need not be long. The art of asking questions which bring out quickly the significant facts is one that should be learned. It is in this respect that the examination of the patient by the specialist differs most from that by the inexperienced practitioner.

There are few diseases that demand more care in history-taking than pulmonary tuberculosis. How the history will be composed and what conclusions will be reached will depend here as elsewhere upon the physician's conception of the pathology of the disease. A peculiarly unprofitable task, it seems to the writer, is the effort to identify the source of infection in the pulmonary tuberculosis of adults, in cases in which there is no family history of tuberculosis.

STATUS PRAESENS

After recording the anamnesis, the next step is the examination into the patient's present condition, to be determined by inspection, palpation, percussion, and auscultation. To the record of such examination should be added the data obained from laboratory analysis of sputum, urine, blood, etc.; also the x-ray findings, if available.

The beginner should bear in mind the fact that his duty to his patient is not discharged when he has determined the existence or non-existence of the malady for which the patient consults him. If, for example, the patient has no active tuberculosis, what is the cause of the symptoms which brought him to the office? This is an aspect of treatment which is too much neglected. The patient may have some serious disease which he does not suspect. It is largely this neglect to get at the bottom facts which leads to the deplorable condition that the autopsy often reveals disease of the viscera, not suspected during life, as the real, or important, contributing cause of death. "The trouble with coming to a hospital," Sir William Osler once remarked, "is that the patient finds things the matter with him which he did not know of." The practitioner, in his review of his patient's case, should emulate the thoroughness of the best hospital methods.

It is a common practice to use blank forms for recording the present condition, on which is printed a list of the various matters which need record in every case, such as the condition with respect to the urine, the intestines, the weight, the general health, etc.; or a list of the physical

signs usually to be observed in a disease with which the physician has much to do—as, for example, pulmonary tuberculosis—or a combination of the two. Such lists are of advantage in reminding the inexperienced to record systematically data which he might otherwise forget to note. The objection to printed forms is that, if sufficient space is allowed for all possible entries, the size and the number of sheets necessary make too bulky a record. If, on the other hand, the entries are crowded, the record of especially important findings overflows to neighboring rubrics, to the confusion of the record; or, what is worse, the recorder, for lack of space, slights the data of which a complete record is most essential.

This objection applies with less force to the lists of physical signs in a disease like tuberculosis. Many forms are available, with printed outlines of the chest, upon which outlines the physical signs and, at the same time, their location may be recorded. Abbreviations and arbitrary figures are employed to save time. In institution work such forms are in common use; the various recorders here have the opportunity to learn and to use readily the signs and abbreviations required, which unfortunately differ considerably in the diverse forms that have been proposed.

Whether the general practitioner is benefited by the use of more general forms, the writer doubts, although much depends upon the nature of his practice. The use of forms undoubtedly tends to encourage routine records, and routine is precisely what should be most carefully avoided.

CASE HISTORIES

Fully as important as the anamnesis and the record of present condition is the recording of the progress of the case under treatment. Two objects are to be sought. First, the aim should be to make notes from time to time which, combined, shall give a consecutive and clear, though brief, history of the case. Careful records of this kind are especially important in institutions in which one medical man may succeed another in the charge of the same case; and there is no part of such work that is more frequently neglected or more badly done. Second, it is desirable to have a record of signs and symptoms which, though perhaps obscure or slight, may point to the development of a complication. One who is not careful to note unexplained signs and symptoms will sometimes regret that he has no record of the exact date of the incipiency of complications which in time prove important or interesting.

CHAPTER II

DIAGNOSTIC PROCEDURES AS APPLIED TO DISEASES OF THE LUNGS

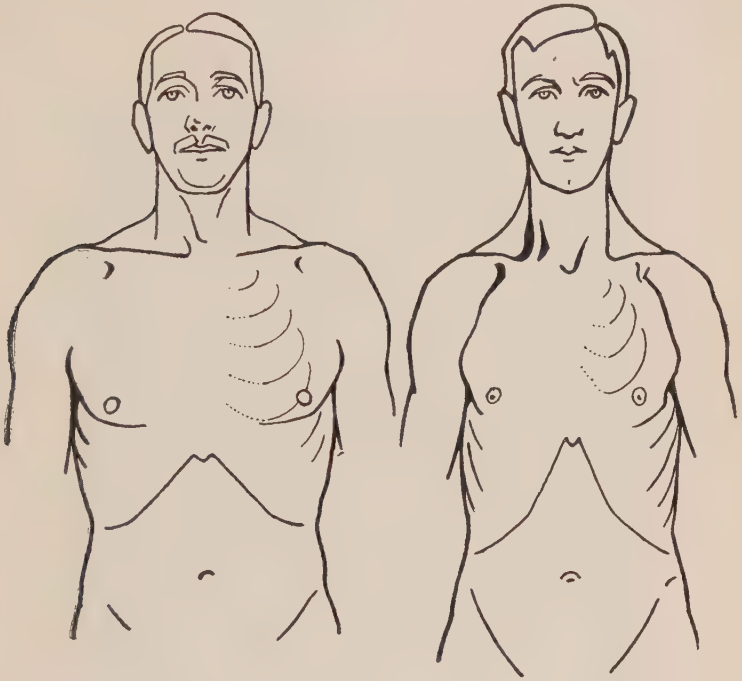
INSPECTION

The physical examination is now undertaken. Patients appreciate the thoughtfulness for their comfort which leads the physician to make such observations as are possible before they are required to strip. The head can of course be examined in advance of this. A rapid glance decides as to corneal opacities and other superficial affections of the eye and of the lids, irregularities of the pupil, etc. Unilateral dilation of the pupil and the Argyll-Robertson pupil should be looked for. It is perhaps not necessary to use the ophthalmoscope in the routine examination of diseases of the chest, but it is a decided advantage to have command of this instrument so that it can be employed when needed. The ears are given such special attention as the anamnesis may have indicated to be necessary, and the nose and throat are examined with especial reference to mouth breathing and enlarged tonsils. The teeth and the mouth should not be overlooked. Nothing affords a better idea as to the state of the general health than the condition of the mucous membrane of the mouth. The neck should be scrutinized for scars or swellings that are indicative of past or present implication of the cervical glands in what are probably tuberculous processes.

The normal infantile thorax has a nearly circular cross-section. With normal development the thorax of the adult comes to have an elliptical cross-section, the longer diameter of the ellipse extending in a frontal direction from side to side. The thorax, however, should have depth; the dorsoventral diameter should not be greatly diminished, as compared with the frontal diameter. When the correct proportion between these two principal diameters is maintained, the length of the thorax is not great.

Developmental or congenital anomalies lead to variation from the normal proportions of the thorax in two ways. First, the dorsoventral diameter is diminished. The frontal diameter may be, and usually is, also absolutely diminished, but is too long relatively to the diameter from side to side; the chest is abnormally flat and shallow. To compensate for

the deficient area of the cross-section, the thorax shoots up and becomes too long, as well as too flat and too narrow. The ribs are more sharply inclined downwards, and the junction of the costal cartilages with the



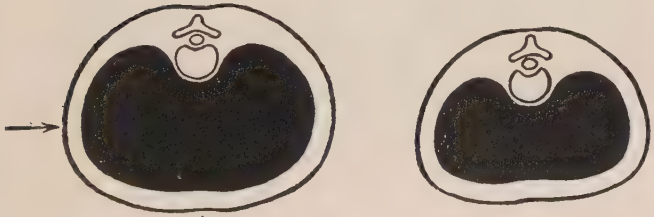


FIG. 1. FIG. 2.

Fig. 1.—Normal thorax. Fig. 2.—Paralytic thorax. (Külbs, "Leitfaden der medizinischen-klinischen Propädeutik," J. Springer.)

sternum is at a more acute angle. By reason of this the costal arch and the crests of the ilium are abnormally approximated. Since the distance between the first rib and the diaphragm is abnormally great, the heart, which is hung on the large vessels—the trachea and the superior aperture

of the thorax—is deprived of support below and becomes a pendulous or "drop" heart, *cor pendulum*. This type of thorax is called the "paralytic thorax," on the assumption that its peculiarities are due to a weakness of the intercostal muscles and of the muscles of the shoulder-girdle. The intercostal spaces are abnormally wide in this type.

In the second type the ventrodorsal diameter is relatively increased

Fig. 3.—Pigeon-breast. Sternum projects forward. Dorsoventral diameter increased. (Collection of Dr. C. N. B. Camac.)

over the frontal diameter; the chest is abnormally narrow, but relatively deep. This approaches the type of the chest of quadrupeds, in which the cross-section of the chest is heart-shaped; its longest diameter is the dorsoventral. Sometimes the human chest is frankly of the quadrupedal type, with projecting sternum, pigeon-breast, *pectus carinatum*. More frequently the chest is flat in front but narrow, the sides of the thorax being also flattened and converging somewhat as they pass forward. This is the "slab-sided" chest. In this type while the anterior tho-

racic wall has an approximately normal extension in length, the part of the spine below the 12th dorsal vertebra is absolutely too long. According to Kraus [1] the measurements of a normal man are: spine, from vertebra prominens to end of sacrum, 67 cm.; dorsal spine, 37 cm.; lumbar spine, 30 cm.; but in two narrow-chested men of about the same height the measurements were 67, 31, and 36 cm., and 66, 29, and 37 cm., respectively. In this, the enteroptotic type, the thorax is often judged from its narrowness to be slighter than it really is. When viewed from the side, it is seen to have considerable depth (that is, a long dorsoventral diameter).

The chief difficulties are now encountered below the diaphragm. The abdominal cavity is disproportionately long; the viscera do not fill it comfortably. This is especially true if there is no fat in the abdomen. The erect position puts the abdomen under more or less tension. The individual prefers a stooped carriage; a kyphosis without a corresponding lordosis is, consequently, often developed. When the subject sits or lies, he is more comfortable if the body is bent forward, thus unconsciously diminishing the length and volume of the abdomen and bringing the abdominal viscera into closer contact with the diaphragm. Enteroptosis is very largely a disease of the diaphragm and of the abdominal wall. (Wenckebach.) Unless the abdominal tonus is good, there is danger that the abdominal viscera will not act efficiently to crowd up the diaphragm in expiration. The diaphragm, not being pushed up in expiration, cannot contract efficiently in inspiration. Remaining flat, its contractions would simply serve to narrow the costal angle. The result is too often a more or less permanent flattening of the diaphragm, with relative respiratory disuse of its musculature. Whereas in the paralytic thorax the difficulty was a too great height of the base of the heart above the diaphragm, in enteroptosis the difficulty chiefly is that the stand of the diaphragm is too low. The result in either case is the drop heart.

The enteroptotic type of chest should be clearly distinguished from that of the paralytic thorax. The enteroptotic individual has troubles of his own, arthritic and circulatory, but especial proneness to tuberculosis is not one of them. If tuberculosis does occur in persons of this build, it is likely to be chronic and capable of relatively easy arrest, as it often is in arthritic subjects. Stiller has described what is practically equivalent to the paralytic thorax—a poorly developed, long, narrow, and relatively shallow thorax, the peculiarities of which he ascribes to *asthenia universalis congenita*, that is, to general congenital weakness. The im-

[1] Brugsch and Schittenhelm, "Lehrbuch klinischer Untersuchungsmethoden," Berlin and Vienna, 1911.

portant question is whether the paralytic thorax antedates tuberculosis and is in a sense its cause, or whether the poor development of the chest is the effect, not the cause, of an early tuberculosis. Though the former view has been the prevailing one, in consideration of the great frequency of tuberculosis in early life there would seem to be little doubt that tuberculosis is in part responsible for the defective development of the skeleton of the trunk. But whatever the rôle played by tuberculosis and general weakness, there is no question that the various deformities mentioned are also to be brought into connection with rachitis.

In judging of the health and stamina of an individual, too much importance should not be given to the shape of the thorax. For it is to be remembered that, though rachitis may have left its indelible mark upon the skeleton, the disease is a self-limited one, and that many children with long and narrow, as well as with enteroptotic chests become strong and vigorous men, and that an abnormally long and narrow chest may have so abnormal a length that the cubic contents equal those of a more athletic chest.

The "rachitic rosary" consists in a series of enlargements at the junctions of the bony and cartilaginous portions of the ribs. The rosary, as its name implies, is due to rickets. The funnel-breast, the opposite of the pigeon-breast, is said to be of congenital origin. The shoemakers' breast, a depression resembling the funnel-breast, is due to the practice of pressing the implements of the craft against the chest. The depression, however, involves the lowest part of the gladiolus and the xiphoid appendix; that of the funnel-breast is situated at a somewhat higher level. Other more or less irregular and atypical elevations and depressions of the ribs are frequently seen, which are for the most due to rickets.

All important deviations from the normal in the shape of the chest should be noted in the patient's history, since the abnormal shape of the bones affects the percussion-note, increase in the arching of the ribs producing dullness, and flattening, greater resonance. Localized dullness not thus accounted for would be considered, by one who reads the record or studies the diagrams without seeing the patient, a sign of disease of the lung.

The relative height of the shoulders should be noted, as well as the presence and degree of the scoliosis which is the chief cause of the difference in height. Lagging—that is, delay in the rising of one side of the chest in inspiration—and the kindred condition, Kuthy's "acromial symptom," [2] a delay in the rising of the shoulder of the affected side

[2] Kuthy und Wolff-Eisner, "Die Prognosenstellung bei der Lungentuberkulose." Berlin und Wien, 1914.

during inspiration—are due to some condition that impairs the expansibility of the lung.

A long list of the rarer conditions determinable by inspection might be given but would be unprofitable. Usually they are quickly discovered.

The triumphs of the expert who determines at a glance such conditions as, for example, the barely perceptible pulsations of an aortic aneurism, or the shape of the liver that betrays amebic abscess, make a great impression upon the student. For this reason inspection is magnified in his mind, as a short cut to a difficult diagnosis. But the expert whose decision seems so instinctive has acquired his skill by long and faithful practice of auscultation and percussion, to which at first, save for an occasional spectacular finding due to a quick eye, inspection comes in largely as confirmatory evidence. Many things determinable by inspection are better determined by other means. It is, therefore, a waste of time for the beginner to make a long scrutiny of the surface of the patient's body at the outset of his examination. He should see quickly what is to be seen, and resort again if need be to inspection, to confirm what he finds by the less showy, but for him more reliable methods of percussion and auscultation.

Normal Weight.—Weight tables for adults are constructed by measuring the height of a large number of men and women and comparing their average height with the average weight of males and females separately for each year of adult life, up to old age. All that such tables can possibly show is the *average* weight. But as a rule the users of the tables take a further step, and call the average weight for height and age the normal weight of the individual in question. It is said that such a person is so many pounds above or below the *normal* weight, and, if he is below the average, the effort is made to raise his weight to at least the average weight of his class. That this theory is not to be relied upon is shown by the fact that it has recently been noted in life insurance circles that persons below the average weight of their height class have a better expectation of life than those at or above the average weight in the same class.

The normal weight of an individual is the weight of his skeleton, viscera, muscles, blood, etc., plus some fat. The normal person has a small *panniculus adiposus;* he should not be emaciated, nor should he have a considerable accumulation of fat. Of the two extremes, too little fat within limits is probably safer than too much fat. The individual who is below the average weight of his class is too often encouraged to excessive eating in order to reach the average. If he chances to be stockily built, such a course might probably be defended if not too long continued.

But he is usually long and slim of chest. A man of such a build is expected to reach an average which was arrived at, doubtless, from a collection of individuals many of whom had deeper chests and broader frames. The skeleton of the adult is fixed; he cannot broaden it to any material extent. The only way in which he can add to his weight is by increasing the bulk of his fat and muscles. Practically, the fat alone is increased if the weight of an invalid increases. To reach "normal" weight such a person puts on, or tries to put on, an amount of fat which for him is abnormal.

A person who gains weight without great difficulty shows in so doing that he has no grave disturbance of digestion or metabolism. If we make a distinction between "tuberculous" and "consumptive," he is not a consumptive—a fact which can generally be established without resort to the scales. Of course there can be no objection to putting on a little more weight when the patient gives up work and takes a rest. But a great gain in weight due to a long course of excess in eating is no proof that the patient will not in time become a consumptive; in fact, so far as the gain has been made at the expense of the integrity of stomach and intestine—not to mention the other evils of stuffing—its effect is prejudicial in the long run as respects chronic wasting from tuberculosis and many other causes of death.

The excuse for bringing this subject forward in a handbook of physical diagnosis lies in the fact that inspection, not weighing and the consultation of tables, is the logical and easy way to determine whether the patient is above or below his normal weight. The cardinal point to be remembered with reference to feeding, it may be remarked in passing, is that no one should ever be encouraged or permitted to eat more than he can properly digest.

It often happens, in the case of subjects of slim build (who not infrequently have an abnormally hearty appetite), that increase to normal weight is most advantageously secured by decreasing the consumption of food.

PALPATION

The employment of palpation in the study of tactile fremitus in diseases of the circulatory and respiratory organs, and of the pulsations, normal and abnormal, of heart and arteries, as well as of the transmission of the voice in health and in disease, is considered elsewhere. Palpation is largely resorted to as a part of the general examination in investigating

the great variety of swellings (tumors, etc.), points of tenderness and the like, which may be encountered in the individual case. Palpation is preferable to inspection for the accurate determination of the condition of the superficial lymph glands. Slight enlargements of these glands are often of considerable significance. The supraclavicular gland, or glands, will not infrequently be found to be enlarged. In such case the presumption is that the enlargement is due to tuberculosis, and it will often be found to accompany a tuberculous process in the lung of the same side. Enlarged occipital and cubital glands create a suspicion of syphilis. Enlargement of cervical glands is often, but by no means always, of tuberculous origin. The connection of cervical adenitis with infection in childhood through milk with the bovine type of the tubercle bacillus is a subject to which attention may well be directed in the anamnesis. The thyroid gland should, of course, be palpated with care.

Increased tension of the thoracic muscles in relatively early pulmonary disease, followed by atrophy in the chronic stages, has been described. The subject is an interesting one, but the information derived from palpation is neither as definite nor as reliable, as that obtained by auscultation and percussion. Palpation of the thoracic muscles in early tuberculosis cannot be said to have established itself as a necessary adjunct in diagnosis.

Deviation of the trachea is one of the manifestations of the dislocation of the mediastinal organs due to unilateral changes in volume of the contents of the thorax, which is discussed in Chapter III. Webb recommends bimanual palpation for its detection when it is not manifest on inspection.[3] The fingers of both hands are laid upon the shoulders, palmar surface down, and the thumbs are pressed in at the sides of the trachea. It is then easy to detect curvature of the trachea outward on the side of the deviation and an abnormal lack of resistance on the other side, the tip of the thumb appearing to enter a hollow space.

PERCUSSION

Percussion may be defined as the art of eliciting sound by which is determined the condition of the organs and tissues of the human body, with respect to the presence of air, and the boundaries of solid organs which border upon air-containing tissues.

The percussion-note may be high or low as to pitch; loud or weak as to loudness; long or short as to duration.

[3] Webb, Forster and Gilbert, "Trachea Position," *J. Am. M. Ass., Chicago,* 1915, LXV, 1017.

The sound conditions determined by percussion are: resonance, dullness, and flatness.

Resonance is divided into non-tympanitic, or normal, lung-resonance, and tympanitic resonance.

Normal resonance, or the note of the normal lung, is the note obtained over the greater part of the healthy lung. It depends upon the vibration of air-cells and is therefore called vesicular resonance. The note of normal resonance is low in pitch and of long duration.

The low-pitched tones which constitute the preponderating element of the pulmonary resonance are due to the vibration in unison of the air and solid portions of subdivisions of the lung, or of the whole lung. The air-cells suppres in large measure the higher tones that proceed from the bronchi.

Hyperresonance is an exaggerated pulmonary resonance, without material alteration of its characteristics. It is the percussive equivalent of puerile or exaggerated breathing, being usually found in the healthy lung which functionates more strongly than usual on account of defective expansibility of the other lung. If the lung is thereby increased in size, the pitch of the percussion-note is lowered, because the expanded chest transmits the deep tones of the lung more distinctly.

Tympanitic resonance is the resonance of a cavity, or group of cavities (including in the latter, relaxed lung), which contain air, and the walls of which are elastic and free to vibrate. Tympanitic sound is really a noise made up of many more or less discordant tones; but the prominence of the fundamental tone of the cavity with its harmonics gives the note a more musical character than that of the normal lung. It is normal over the stomach and the intestine, but in the lung occurs normally only in Traube's semilunar space, to which it is transmitted from the stomach (see p. 112). The pitch of the note of tympanitic cavities depends chiefly upon the size of the body of air contained within the cavity or group of cavities.

Dullness may be defined as the impairment of resonance, the normal pulmonary resonance being taken as the standard.

There may be normal local variations from the typical resonance of the lungs shown first, by a shorter duration of the percussion-note. The presence of an airless organ, as the heart, behind the part of the lung percussed shortens the duration of the percussion-note; the air-bearing mass thrown into vibration is smaller than the mass that furnishes the normal vibrations, and its vibrations therefore die out more quickly. The pitch of the percussion-note remains unchanged unless the lung-mass percussed is very thin. Second, these variations are shown by apparent

raising of the pitch. In locations where the number of air-cells normally interposed between the bronchi and the chest wall is materially less than the normal average number, the bronchial sounds will be less completely suppressed, and the note will consequently seem to be higher in pitch; that is, the higher-pitched sounds will become more conspicuous; the deeper tones are, however, still present. Third, by a change of quality. The note over the base of the left lung anteriorly has normally a more or less tympanitic quality because of the proximity of the stomach.

According to the definition given, slight dullness is normally present over portions of the apex of the right upper lobe and of the interscapular space, as well as over the parts of the lung bordering on the heart and liver.

Dullness is normal over certain parts of the body that do not contain air, as the liver. The note here is dull rather than flat, because it contains a resonance due to the ribs as a portion of a resonator and to the proximity of air-containing organs. Where no resonator is near to reinforce the sound, percussion gives flatness instead of dullness over airless organs. The term flatness indicates the complete absence of resonance. As thus defined, flatness is rare; for there are almost always resonant bodies within the reach of the sound waves produced by percussion.

Abnormal Resonance, Dullness, and Flatness.—Tympanitic resonance is usually present in pneumothorax if the tension of the air permits. When the air of the pneumothorax is under high positive pressure, the note is not tympanitic. Tympanitic resonance is frequent over superficial cavities of the lung. It occurs over portions of the lung relaxed by disease, as over some tuberculous lesions and above pleural effusions; also when the air-cells contain both liquid and air, as in pulmonary edema. When solidification of a portion of the lung permits the force of percussion to be transmitted directly to a cavity, either normal—as a bronchus—or abnormal, which lies beneath, the percussion-note, instead of being dull, may be tympanitic.

Tympanitic sound is not sharply distinguished from atympanitic. Biermer has described an intermediate stage under the name of "box-tone" (*Schachtelton*). This is found when the tension of the lung-tissue is not abolished, but is impaired by overdistention, as in emphysema or, more acutely, in attacks of bronchial asthma. The tone has not the more or less pure musical quality of tympanitic resonance, but resembles the sound produced by striking an empty bandbox. It is still more like the sound of percussion of a cushion stuffed with hair or feathers (*Kissenton*), in which the structure in some respects resembles that of the re-

laxed lung, being a cavity, made up of numerous cavities, which resounds as a whole.

In abnormal dullness there is usually a relative, or complete, absence of low-pitched, and a relative increase of high-pitched sound waves. The higher tones are heard more distinctly, both because of the relative absence of low tones and because the high tones are absolutely louder. The percussion-note is, therefore, of higher pitch and of shorter duration than over a normal lung, high-pitched tones dying out more quickly than low-pitched tones.

Causes of impairment of resonance are of two classes. First, causes that interfere with the vibrations *en masse* of the part of the lung percussed; as fibrosis, pleural adhesions, deep foci beneath healthy lung. Second, local causes; any morbid process that destroys air-cells, or deprives them of air, either entirely—pneumonic infiltration, caseation, atelectasis, massive fibrosis, abscess, tumor, gangrene, aneurism—or partially (disseminated fibrosis).

In the first class, the duration of the low-pitched sounds will be less and there will be more or less increase in high-pitched sounds, but the percussion-note will not differ greatly from normal. In the second class, the low-pitched tones will be of shorter duration and weaker, or entirely absent. The higher-pitched tones will be relatively more pronounced; while, absolutely, they may be well-marked or weak according to the conditions in the individual case.

Except in emphysema and in pneumothorax, the pitch of the percussion-note in disease is always higher than the normal note.

Abnormal flatness is practically confined in lung-conditions to the note usually elicited over massive consolidations and pleural effusions.

Metallic resonance and "cracked-pot" sounds are phenomena encountered occasionally in percussion under special conditions. Metallic sounds arise in large, smooth, air-containing cavities—stomach, large superficial pulmonary cavities, pneumothorax. These very high-pitched tones are really the disharmonic overtones of the fundamental tone of the cavity. They are best elicited by percussing with a rod upon a metal pleximeter (*"Stäbchen" percussion*). (See p. 105.)

If the basal tone of the cavity is brought out by the percussion which elicits metallic sounds, the phenomenon is known as the "amphoric echo." The term amphoric (from *amphora,* a jar) denotes a sound resembling that caused by blowing across the neck of a flask. (See p. 103.) Amphoric resonance may be obtained by percussion of the normal trachea.

The "cracked-pot" sound much resembles the chinking sound produced when the hands loosely clasped are struck against the knee. It is sup-

posed to be due to the escape of air from a nearly closed cavity, with a hissing sound that interrupts the metallic tones of the cavity. A flexible chest and, usually, strong percussion are essential for its production. It is most frequent over large and superficial cavities of the lung, but may occasionally be heard over the chest of the healthy child.

FIG. 4.—Immediate percussion. (Külbs, "Leitfaden der medizinischen-klinischen Propädeutik," J. Springer.)

The Practice of Percussion.—The Salernitan School of Medicine taught percussion of the abdomen, to distinguish between dropsy and tympanites. Auenbrugger, who discovered percussion of the lung practised it by striking the chest directly with one or more fingers of the gloved hand. This immediate percussion is practically not used, although Geigel says that in certain cases it has advantages over mediate percussion. One can orient himself well, he says, as to the air-content of the whole apex of a man, if he is not too fat, by one blow over the clavicle, and immediate percussion of the lower lobes over the back reveals by one blow on right and left, a difference in the air-content—for example, a beginning hypostasis—not always easily demonstrated by mediate percussion.[4]

In mediate percussion the blow is delivered upon a pleximeter instead of upon the chest wall directly. The instrument known as a pleximeter is a plate of varying size and composed of various materials, usually hard rubber or ivory. The percussion is performed by means of a percussion hammer, the face of which should be covered with soft rubber to deaden as far as possible the sounds which result from striking together two hard surfaces. Extraneous sounds are not altogether prevented by this device, but with practice what remains of them can be overlooked. It is possible to obtain excellent results with this instrument in the hands of an expert; but it has the disadvantages of adding to the armamentarium, and of the fact that the pleximeter is sometimes too wide to enter a narrow

[4] R. Geigel, "Leitfaden der diagnostischen Akustik," Stuttgart 1908.

intercostal space. The finger is more generally used as a plexímeter; in fact, the finger is usually spoken of as a plexímeter. The word plexímeter in what follows is to be considered as including the finger used as such, the blow being given by a finger of the other hand as a percussion hammer.

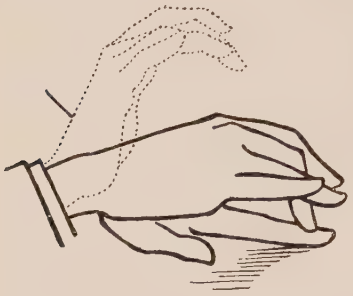


Fig. 5.

Fig. 6.

Fig. 5.—Strong percussion. Fig. 6.—Weak percussion. (Külbs, "Leitfaden der medizinischen-klinischen Propädeutik," J. Springer.)

In immediate percussion much of the effect of the blow is lost, if the superficial parts are thick, from the absorption of the vibrations by them; it requires a strong blow to elicit pulmonary resonance. Over a hard body like the skull mediate affords no advantage over immediate percussion; but in percussion of the thorax, the effect of pressure with the plexímeter is to condense the superficial soft parts and to bring them more closely in contact acoustically with the ribs and with the tissues beneath the ribs. This affords a hard exterior surface, so that the force of the blow will not be deadened by the softness and yielding character of the skin and muscles. The plexímeter becomes acoustically a part of the chest when it is pressed firmly upon it. The sound of the blow is transmitted by it to the chest wall, and the commotion of the blow also is transmitted; the thorax is caused to vibrate by the direct force of the percussion, as well as by the sound waves which proceed from the plexímeter. The firmer the pressure, the more completely are the vibrations of the plexímeter communicated to the underlying parts, and the more are these parts condensed. Hence it is said that increase of pressure upon the plexímeter is equivalent to increase of force in the percussion-stroke. But increase of pressure results in a wider dissemination of the sound of percussion over the thorax. The rib, though perhaps not actually struck, is always very near. As an elastic and relatively dense body, it transmits sound with greater facility than do the soft parts. The direction of the

layers of the soft superficial tissues bounded laterally by the ribs is such that the sounds transmitted by them follow the course of the ribs. The vibrations of the ribs and of the soft parts are freely communicated from one to the other, and the sound is carried by both—but in larger measure by the ribs—in a direction preponderatingly outward, then backward and upward, in percussion anteriorly; forward and downward when the back of the chest is percussed. The sound is given off from both bone and soft parts in its passage around the chest to the lung encircled by the thoracic wall.

The stronger the blow and the greater the pressure of the pleximeter, the more extensive the portions of the thorax that vibrate and the more certainly the resonance produced is not localized to the part percussed. There is rarely any advantage in strong pressure upon the pleximeter, but it should always be brought into complete and firm apposition with the underlying parts of the chest, contact with the ribs being avoided as far as possible.

In percussing, it is desirable to stand directly in front of the subject. Experience shows that it is difficult to judge of a percussion-note if one stands, for example, opposite the other side of the body; and that the best results are obtained by bringing the ear as close as possible to the point of percussion, and directly before it. Geigel says that percussion celebrates its triumphs when it elicits sound waves of local origin. That is to say what is desired in the study of morbid conditions of the lungs is to ascertain to what degree sounds differing in character from the general lung-resonance are obtainable locally. These sounds are at best relatively weak. Since the loudness of sounds diminishes as the square of the distance, if one stands twice as far away as is necessary, he will obtain only one-fourth of the sound that might have been audible. If the object of percussion is simply to determine whether or not the resonance of one side of the chest differs from that of the other side, the exact position of the observer is of less importance.

The observer standing directly before the part percussed, the pleximeter is pressed firmly against the chest wall in an intercostal space. The percussion-blow should be delivered squarely upon the centre of the back of the second phalanx of the pleximeter-finger (usually the middle finger of the left hand), in a direction vertical to the surface percussed. The nail of the percussing finger should be kept trimmed so that the top of the terminal phalanx may be used as the face of the hammer. Theoretically, it has been held that the more rapidly the percussing finger springs back, the more localized is the effect of the blow, and accordingly the direction is given to strike a short, sharp blow from the wrist, with quick recov-

ery, the technic being that used in striking a staccato note on the piano. It is much more difficult, however, to strike the plexímeter accurately when the percussing finger passes through a wide range of movement. According to the most recent investigations, the most favorable conditions for the observation of lung-sounds will be present when the time of contact is long enough for the vibrations of the chest wall to be damped by the hammer, but not so long that the more sluggish vibrations of the lung also will be damped.

Some successful diagnosticians raise the finger but little after the percussion-stroke, and give what might be described as a pushing blow without attention to quick recovery. It is well to cultivate a correct technic, but it is of chief importance to deliver each blow at the proper place, in the proper direction, and in exactly the same way each time, which process is facilitated by not raising the finger far after the stroke. The force of the blow should generally be sufficient to produce the resonant lung-note, not strongly but distinctly. With strong percussion there is danger that the high-pitched sounds from comparatively small lesions will be lost in the general resonance of the lung. Dullness that persists though the percussion be strong proceeds from lesions of considerable size, a point which may be utilized in determining the extent of the disease process. The danger of very light percussion is that a blow ineffective in calling forth the perhaps existent normal resonance, on account of its slight force or because of imperfect apposition of the plexímeter, may be misinterpreted to mean abnormal dullness; the lighter the percussion and the less the pressure of the plexímeter, the greater the chances of such an error.

The lung-note having been obtained, it should be analyzed. It consists of sounds of various pitch, which the student should learn to recognize. The highest sound is that of the plexímeter. This is very distinct with a hard rubber plexímeter, but only faintly audible when the finger is struck and the lung-note elicited. It is well to study this sound by percussing the finger pressed firmly against the concha and over the meatus, when it will be found surprisingly loud. The lowest sound audible in the percussion-note is that of the fundamental tone of the thorax. With this are mingled higher tones from the bronchi, in greater or less proportion according to the location and the health of the parts percussed, which are not always easily distinguished, but which have their part in producing the quality of the lung-note. When the bronchial element of the sound is unduly prominent, the pitch of the note will be raised.

One should pay close attention to the pitch. Especially to be noted are the low tones of the percussion-note. If the note is found to be higher,

this really means the diminution or absence of the low tones, which permits the higher tones to be heard more distinctly. Being higher, the note over the diseased part dies out more quickly. Hence a high-pitched percussion-note is sometimes spoken of as shorter or shortened. The duration of the sound should be carefully studied. One whose ear is deficient in appreciation of pitch can still do good work by concentrating his attention upon the duration of the percussion-sound. But a good musical ear does not guarantee success in discriminating individual tones in a composite sound. Concentration of the attention and considerable practice are necessary for this.

Having by this study learned to appreciate the characters of the percussion-note, the next step is to practice concentration of the attention upon the recollection of the sound heard, so that it may be compared with the sound of the succeeding blow. This is very easy as between absolute heart-dullness and lung-resonance for example, but difficult in the determination of the exact boundaries of relative heart-dullness.

The secret of success in percussion depends upon (1) accuracy and uniformity in the strokes; (2) analysis of the sound produced; (3) recollection of the sound produced so that it can be compared with the sound resulting from the next stroke. The student should practice systematically upon the healthy subject until he acquires the habit of percussion. Percussion with short, sharp blows and with a pushing stroke, with slight and with considerable force, may yield results apparently of equal value in the hands of experts, because each individual percusses invariably in the same way and has learned to analyze his results. One, for example, percusses lightly and avoids calling forth strong lung-resonance; another, with a stronger blow, learns to disregard the deep tones of the general lung-resonance and to concentrate his attention upon the higher-pitched tones transmitted from the lesion. It is, therefore, of prime importance to acquire a habit of percussion—an elegant method of course, if possible, but, at all events, one that will always furnish strictly comparable results.

In a general survey of the chest by means of percussion, the procedure is more conveniently carried out if the patient stands. In office practice it is customary to have the patient sit upon a revolving stool, the examiner also being seated. In the case of weak patients, it is possible to make a fairly satisfactory examination of the front of the chest while the patient lies upon his back, raising him to a sitting position when the back is examined.

In acute illness, as pneumonia, the part complained of will naturally be first examined. The examination of the lungs for the presence of pulmo-

nary tuberculosis is generally begun with the apices, for the reason that the upper parts of the lungs are usually first affected with manifest tuberculosis. It is generally advised to compare each part percussed with the other side at the same level. But both apices often show abnormalities. In cases where this is suspected, it is necessary to compare the part percussed with another portion of the lung at a lower level. No examination is complete without a careful analysis of the percussion-note of the suspected area, a study of the high-pitched and low-pitched components of percussion-sound separately. The student who practices such analysis will find that with increasing proficiency comparison with the opposite side becomes less and less necessary. He is now able to discriminate between healthy and diseased tissues because he knows the percussive characteristics of each.

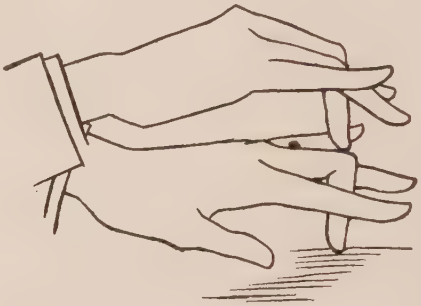


Fig. 7.—Illustrating the position of the finger in orthopercussion. (Külbs, "Leitfaden der medizinischen-klinischen Propädeutik," J. Springer.)

The existence and location of the lesion being determined, this portion of the lung is then studied intensively, with the aid of auscultation, palpation, etc. It is often the practice to study the chest only as a whole; that is, to percuss it and to auscultate it everywhere, front and back, once for all. But if the examiner is content with this examination and attempts to combine all of these findings into a complete diagnosis, there is danger that some essentials may be forgotten or overlooked. The writer is accustomed to make a rapid preliminary exploration of the chest as a whole, and then to concentrate upon the found lesion in the manner just described. In the case of the very ill patient, little more can often be done than to study intensively the parts accessible, for the reason that the patient cannot be raised or turned from side to side. It is sometimes advisable that percussion of the chest shall begin at the base of the lung and proceed upward. This has the advantage in the great majority of cases of obtaining at the outset the full pulmonary resonance. The examiner finds the strength of percussion that will make this note just

distinctly discernible and if, as he continues upward, he gives that same force to the blow and no more, he will be in the position to detect any slight impairment of resonance that may be present. In doubtful cases it may be well to percuss both upward and downward and to check the results of one method with those of the other.

It is held by many that in light percussion more information is obtained from the sense of touch than from the sound elicited; that, in other words, the examiner detects the lesion by the feeling of increased resistance to the plexímeter-finger. By others it is claimed that this is a delusion, because the method cannot be carried out successfully if extraneous sound prevents the hearing of the percussion-note.

There is no doubt that the determination of the degree to which tissues yield to the blow of percussion, or of the sense of resistance, is as truly an objective means of diagnosis as is the sound of percussion. The method is, however, a more difficult one, if cultivated to the point of solving difficult questions with some certainty. The technical skill employed in perfecting such a method would be more profitably applied in the study of the percussion-sound.

To depend chiefly upon the feeling of resistance rather than upon the sound of percussion is, like dependence upon duration rather than upon pitch of sound, to abandon the best criterion for one of inferior value.

Plesch has described a method which facilitates the percussion of extremely limited areas.[5] The left middle finger is bent at a right angle

Fig. 8.—Finger-finger percussion between the heads of the sternocleidomastoid muscle. Fig. 9.—Percussion with the glass rod. (Goldscheider, Berl. klin. Wchnschr., XLIV.)

at the joint between the first (proximal) and the second phalanx, the second and third phalanges being kept extended. The pulp of the finger tip

[5] Plesch. *Deutsches Arch. f. klin. Med.* 1908, XCIII, 199.

is then pressed upon the point selected and percussion is made close to the distal extremity of the first phalanx. This method, which is known as orthopercussion, is of use chiefly to percuss the apex where it becomes accessible above the clavicle, between the insertions of the sternomastoid muscle. Goldscheider has devised a glass-rod pleximeter for the same purpose of limiting the percussion to a small area, which has the advantage of giving access to localities that cannot well be reached by pleximeters of ordinary pattern. Plesch's method is, however, sufficient for ordinary examinations.

"Threshold" percussion was first introduced by Ewald, but has been developed as a method by Goldscheider.[6] In this method the distinction between resonant and less resonant areas is made by adjusting the force of the percussion-stroke so that it will be inaudible over the dull area, and will become apparent when the resonant area is reached, on the principle that the transition between no sound and a faint sound is more readily perceived than a difference between two faint sounds. It is recommended for use only in percussion of the apex and for the determination of absolute heart-dullness.

Auscultatory Percussion.—Since the walls and the air of a cavity furnishing tympanitic resonance vibrate as a whole, it is impossible to percuss out local differences of sound in stomach and intestines. Moreover, the tympanitic sound is so readily induced under favorable conditions of tension, that percussion of the edges of solid bodies which neighbor upon tympanitically-resounding air-spaces results in the production of tympanitic resonance. Hence "the percussion of liver, heart, and spleen against the lung-resonance may give correct results, but percussion against the tympanitic sound of the intestines shows these organs too small." To map out the boundaries of a solid organ against tympanitic resonance, and to determine the exact boundaries of two airless organs in contact with one another, recourse is had to auscultatory percussion. Either light percussion is made over the organ examined, or a sound is produced by scratching the skin over it with the finger nail. The latter is preferable because easier of performance, the examiner requiring one hand to steady the stethoscope, and also because it is not desirable to call forth tympanitic resonance. Whichever method of producing sound is resorted to, the bell of the stethoscope is placed over the organ, and note is made when a change appears in the transmitted sound as the percussion or scratching is shifted from point to point, approaching and finally passing the boundary sought. So long as the sound is produced over the organ auscultated, it is transmitted to the ear with little or no diminution of

[6] Goldscheider. *Berl. klin. Wchnschr.*, 1907, XLIV, 1267.

its intensity. But when the sound has to pass from another organ to the one auscultated, the fact is indicated by a sudden diminution in its force, due to the loss of sound waves from reflection at the surface of the latter. In practice, the best way is to hold the stethoscope over the organ with the left hand, and with the right to scratch the skin gently at a point without the organ and, gradually approaching in a direction at right angles to its edge, to note when the sound suddenly becomes intensified. In making these observations, it is not necessary to take into account the relative distance of the places scratched from the bell of the stethoscope, for variations in intensity are not due in any noticeable degree to changes in that distance, but rather to passage from one acoustic medium to another, with a sudden gain or loss, as the case may be, in the loudness of the sound.

The Results Obtained by Percussion.—As has already been remarked, it is impossible to percuss out local differences of sound in a body which gives tympanitic resonance, because the body vibrates as a whole. The resonance of the stomach has the same pitch, whether the blow is heavy or light—one cannot even determine the level of liquid within it. A large tympanitically-sounding body has a tone of relatively low pitch; the pitch becomes higher, the smaller the vibrating body. A relaxed lung out of the body gives a tympanitic note. Pieces of lung yield a tympanitic sound, the pitch of which varies with the size of the piece.

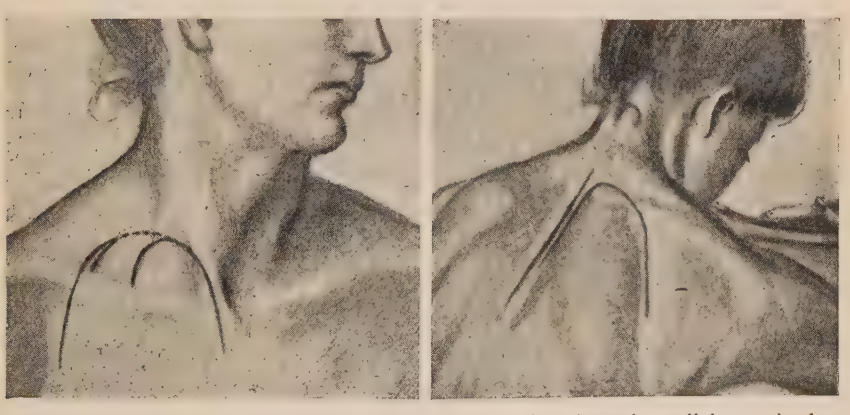


FIG. 10.—Projection of the lung borders on the anterior thoracic wall by sagittal percussion with the glass rod. FIG. 11.—Projection of the lung borders on the posterior thoracic wall. (Goldschieder, Berl. klin. Wchnschr., XLIV.)

Since the lung is a resonator which has a fundamental note of a pitch dependent upon the volume of air contained within it, and since pulmonary resonance resembles tympanitic resonance in that its pitch is not

changed by variations in the force of the percussion, it might be thought that there would be the same difficulty in localizing the effect of percussion over the normal thorax that is experienced over cavities, like the stomach, which give a tympanitic resonance. As a matter of fact, this difficulty does exist when the percussion-blow is forcible and is struck over normal lung. Under these circumstances the resonance is loud and more or less generalized. The only way to escape this result is to resort to light percussion. The lung is not a perfectly homogeneous and very elastic substance, like the air that fills a cavity giving tympanitic resonance. Although its large subdivisions are capable of functioning, in the production of sound, in harmony with the resonator that contains it, its make-up renders it necessarily sluggish in response. While a strong blow produces a resonance which comes from an indefinitely large portion of the lung, a light blow may produce resonance arising in the portion of the lung directly involved by the percussion-blow.

It is, for example, quite possible to percuss out with reasonable accuracy the relative heart-dullness. Using a comparatively light percussion-stroke the sound waves of chest wall and pleximeter, and the more or less localized slower vibrations from the commotion of the blow, penetrate the thin layer of lung which overlies the heart, and set up a special vibration in this mass. The percussion-sound travels at the same time around the ribs, and causes the whole lung to vibrate with the production of the fundamental tone, but so feebly, under the circumstances, that this tone can be disregarded, and the attention fixed upon the shorter sound produced locally. This sound should not be conceived of as the reflection of sound waves that have been sent into the lung from the pleximeter. The low tones of the sound that reach our ears originate in the lung. The sound differs from the general lung-note, however, in that it results from the vibrations of a mass smaller than the subdivisions of the lung which yield the normal thoracic resonance, and therefore dies out more quickly. These vibrations are of the pitch of the fundamental lung-note over the deeper portions of the lung-mass, but become higher and weaker as the absolute heart-dullness is more and more closely approached. Because of the smallness of the mass, they have less energy, and therefore pass out less freely through the chest wall; there is a weakening of the lung-component of the percussion-note on that account.

Now, if the dullness were due chiefly to absorption of the sound waves by the heart, one might expect to be able to outline the true heart-border to a hair's breadth. But if the difference in sound is caused by the fact that the vibrations are those of a smaller resonant body, the sound would change its character gradually—there would be no sharp line of demar-

cation. This is what obtains in the case of the relative heart-dullness. The examiner, it is true, marks with a pencil the exact outline of the heart as he finds it, but the line drawn simply represents the place where a difference in sound was noted with the strength of percussion used. Another examiner, equally skilled, would probably obtain a somewhat different result; even the same examiner, repeating his percussion after effacement of his previous record, would not make exactly the same outlines. The problem is very like that of determining the exact line at which the red glow of the sunset horizon ceases and the blue of the sky appears. That there is this transition is perfectly evident, but no one can fix its boundaries with exactitude.

The presence of a local lesion introduces a new set of conditions. In so far as the part is diseased, it is no longer a sound medium, homogeneous with the remainder of the lung. Fibrosis, infiltrations, and the like deprive the part of some or of all of its air and solidify its structure, so that it conducts high-pitched sounds more readily. Hence a light blow will produce a resonance which is that of the part percussed, and which will differ from the normal by a diminution in low-pitched and an increase in high-pitched sounds. But a strong blow again calls forth the general pulmonary resonance, the vibrations passing through the diseased focus to awaken the general resonance of the thorax beneath; for the consolidated or indurated lung is an excellent conductor of sound.

The large infiltrations of pneumonia, which deprive of air all or the greater part of the lung at the level examined, give, of course, well-marked dullness. Dullness may also be marked over the firm and dense adhesions of a fibrous tuberculous lesion; and in some cases of advanced caseofibrous tuberculosis of the lower lobes, the thickness of the plastic pleural exudate is such that the dullness on percussion is very marked. In these conditions the sound is not only higher and shorter, but also much weaker.

In the deep congestions of incipient lobar pneumonia, as in deep tuberculous foci, the lesion is separated from the pleura by a zone of healthy, or nearly healthy lung. No doubt, the vibrations produced by percussion penetrate to these deep foci; but whether the result of such commotion is returned to the ear as an audible sound is quite another matter. The first result of the blow of percussion is to call forth the note of the thorax-resonator, that is, the thoracic resonance. This sound, it should be remembered, is not the reflected sound of the plexímeter. The plexímeter sound is simply the excitant of resonator action. The sound that we hear, it would be correct to say, originates in the lung. It is not even necessary that an audible sound shall be produced—a *silent* concussion of

the chest may awaken the pulmonary resonance. This pulmonary resonance may be impaired as the result of the presence of the deep lesion; but the inelastic tissues of the disease focus are incapable of vibrations which will yield sound to rival or surpass the sound of the resonator. The result of percussion over a deeply-situated lesion is then, at most, a negative influence upon the sound of the resonator—a diminution of resonance, absence or diminution of the fundamental tone of the thorax, a shorter and higher-pitched note.

Such a result, if there were every reason to suppose a healthy state of the superficial lung, would be, as in the case of a suspected early pneumonia, a valuable finding. It would show that a deep-seated pneumonia was in all probability already present. But in the case of a suspected deep tuberculous focus, the situation would be more complicated; for here it is by no means certain that the superficial lung is healthy. A disseminated thickening of the fibrous tissues, disseminated tubercles, active or obsolete, pleural adhesions that limit vibrations, localized congestions and infiltrations of tuberculous origin, are all very common, and all produce impairment of pulmonary resonance of the same nature as that due to deep lesions. We learn in such conditions that, so far as percussion is concerned, the drum has a changed note: and that is all that can be learned from percussion.

According to Oestreich and De la Camp,[7] the greatest depth to which the percussion-stroke penetrates is 6 cm., from which 2 to 3 cm. must be deducted for the thickness of the superficial soft parts; that is to say, a lesion situated at about the depth of one and a half inches is as deep a lesion as one could hope to detect. But here, of course, the size of the lesion is as important as its depth; and it is a question whether lesions situated at such a depth are ever really detected as such by the aid of percussion alone. There is a vast difference between accurately mapping out a lesion and determining that the region in which a lesion is present has a changed percussion-note. F. von Müller points out that a small strip of lung may completely conceal an aneurism as large as the fist, and states that, in opposition to some modern authors, he has to admit that the deep extension of percussion is slight.[8]

Percussion of the tuberculous lung amounts usually to a comparison of the note of this lung with that of the healthy lung at the same level if possible, and deciding by the pitch, duration, and loudness that a morbid change has occurred. The lesion may be an approximately globular

[7] Oestreich and De la Camp, "Anatomie und physikalische Untersuchungsmethoden," Berlin, 1905.

[8] F. von Müller, *Ztschr. f. ärztl. Fortbild., Jena,* 1912, IX, 417.

focus, yet we record it as going down to the level of a certain rib or vertebra, thus implying an indefinite lateral extension. There is justification for this, because the dissemination of sound along the ribs prevents an accurate fixing of the lateral boundaries of the lesion, except possibly in superficial lesions when extreme care is used. Usually, in small lesions, our results have been a determination, not of the note of the lesion itself, but of the note of the surrounding lung as affected by the presence of the lesion.

The beginner in percussion expects dullness as the sign of a tuberculous lesion—dullness that is brought out by any kind of percussion, so as to be recognized instantly, very much as any sort of stroke reveals the flatness of a large pleural exudate. In practice, however, he learns that the matter is not so simple; he often does not find dullness where he thinks it should be, for well-marked and easily elicited dullness is not a characteristic of many tuberculous lesions. The remark is not uncommonly made that percussion has little or no value in the diagnosis of pulmonary tuberculosis. The reason for this opinion is that the examiner has not standardized his technic. He is not certain how strongly he should percuss, nor, if the first strokes do not obtain resonance, what the meaning of that fact is; he, therefore, increases their force until he has elicited the resonance of the thorax so loudly as to obscure the sounds from the lesion. He then compares the note obtained with that of the other side at the same level: the two notes appear to him alike, and he concludes that there is no change in resonance. Here he has defeated his purpose by committing the mistake of insisting upon obtaining full resonance, and by taking no account of the amount of force that he is employing in so doing.

AUSCULTATION

There are two kinds of auscultation, immediate and mediate. In the former the ear is applied directly to the chest, preferably with the interposition of a towel, not only for the sake of cleanliness, but because the ear tends to adhere to the more or less damp or greasy surface of the skin so that annoying sounds are produced. The head of the examiner should be pressed firmly against the thorax and the free ear should be closed by pressure on the tragus, in order to exclude extraneous sounds. This is the only method of auscultation that our fathers knew. There are manifest objections to it, the most important of which are that certain portions of the thoracic surface cannot be explored by it, notably the

parts above the clavicle and the upper part of the axilla; the undesirably close approximation of the examiner to an unclean or otherwise objectionable individual; considerations of delicacy in the examination of the anterior chest in women; the discomfort and congestion of the head incurred in stooping over a recumbent patient; and the difficulty of accurate localization of sounds. But for the ordinary examination of the male in the sitting posture, and for examination of the back in women, the greater part of these objections do not apply, and the method has certain advantages which should forbid its abandonment.

Succussion for the detection of splashing sounds in pneumothorax is best practised in connection with immediate auscultation. The examiner presses his ear against the chest wall of the patient sitting upright, and rocks the body gently to and fro, his head following the movements of the patient's chest.

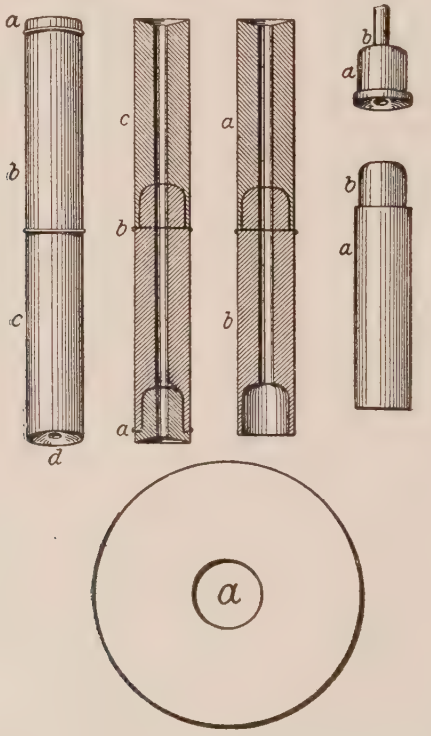


FIG. 12.—Laennec's stethoscope. The cross-section in lower part of figure is the natural size. (Edens, "Lehrbuch der Perkussion und Auskultation," J. Springer.)

Mediate auscultation was first introduced by Laennec. It is performed with the aid of the stethoscope. The early stethoscope consisted

of a solid wooden cylinder, which at a later time was replaced by a wooden tube, partly because a tube vibrates better than a cylinder of the same size and partly to diminish weight.

The monaural (more correctly, uniaural) stethoscope is still used extensively in Europe, but little, if at all, in this country. It transmits sounds more accurately than does the binaural, but is not so convenient to handle, and has the disadvantage that extraneous sounds reach the free ear of the observer.

The special advantage of the monaural stethoscope over the binaural is its better conduction of high-pitched sounds. The precise manner in which this conduction is effected can be better understood after a consideration of the acoustic properties of the external auditory apparatus.

The cartilage of the auricle is of highly elastic material and is so shaped as to collect sound waves and deflect them toward the auditory meatus. It consists of a single piece, but is provided with fissures, which tend to increase its mobility. The cartilage of the external or cartilaginous portion of the meatus is continuous with the cartilage of the auricle, and is likewise rendered extremely movable by vertical fissures (*incisuræ Santorini*). The tragus may be viewed as the prolongation outward of the cartilage of the canal. It serves as a lid to close the meatus when pressed upon. The structure of the external ear in general is manifestly such as to make it responsive to sound vibrations: but at the same time, while providing for the unbroken transmission of sound waves, to prevent or to minimize the proper vibrations of the cartilaginous plate.

The ear is constructed for the reception of vibrations which reach the membrana tympani through the air. Conduction of sounds through solids is of slight importance physiologically. But, however we practice auscultation, the sounds to which we listen are communicated to the ear, directly or indirectly, from the surface of a solid medium, the chest wall. There would seem, therefore, to be no theoretical objection in auscultation to prolonging the transmission of sound through solid media by interposing a solid sound conductor between the observer's ear and the chest wall. This was done by Laennec in the early stethoscope which, as has been stated, consisted of a solid wooden cylinder. This material is not satisfactory for the conduction of low-pitched sound, but it transmits sounds arising in other solid media very perfectly: the high-pitched metallic ringing sound of the watch, for example, is conducted with no apparent loss, if the watch is pressed against one end of the cylinder and the other end rests upon the tragus. In this experiment the conduction of sound has been through solid media, including the tragus. The fact that pressure of the cylinder upon the tragus closes the meatus seems to

make no difference, but from this fact the conclusion should not be drawn that there is conduction through solid media all the way to the inner ear. Conduction does not take place through the bones of the skull. If it did, we should be able to hear the ticking of the watch when it is placed above, behind, or below the auricle which is not the case. Only when the watch is crowded into the space between the meatus and the jaw, where it presses against the external auditory meatus, are we able to perceive the watch sounds, including the metallic ringing. Since the inner portion of the auditory meatus passes through bone, a substance which is not a good transmitter of watch sounds, it is probable that the vibrations imparted to its cartilaginous portion are made audible as sound, by being transmitted to the air of the meatus. From these observations we conclude that through whatever sound media waves of sound are conveyed to the head, they must reach the eardrum by aërial conduction, in order to be appreciated as sound.

FIG. 13.—Monaural stethoscopes after Skoda, Traube and Bass. (Edens, "Lehrbuch der Perkussion und Auskultation," J. Springer.)

That the air within the monaural stethoscope conducts sound is shown by an experiment of Martini.[9] He cut a stethoscope in two transversely, and connected the two halves by means of a short rubber tube. This practically abolished conduction through its walls, but breath-sounds could still be heard through the instrument. According to Martini, if the air-containing space of a monaural stethoscope is cylindrical in shape, not over 12 cm. long, with a lumen not less than 4 mm. in diameter, the instrument may be said to be capable of functionating correctly in the transmission of sound.

The binaural stethoscope, as commonly used, consists of metal ear-tubes, with tips of hard rubber for insertion into the meatus, usually

[9] Paul Martini, *Ztschr. f. Biol. München u. Leipz,* LXXI, 117.

united below by a movable joint, or rigidly, and connected at their lower ends by tubes of soft rubber with a hard rubber bell. Since some fail to understand the possibilities of this instrument and the precautions necessary to realize them, its structure and use will be considered in some detail.

It is indispensable, in the first place, that the stethoscope fit the ears. Many stethoscopes are too small and, particularly, have too small ear-tips. The ear-tips should fit the meatus comfortably. If they are too small, the observer is able to carry on a conversation with ease, hear the clock tick, etc., without removing the instrument. This should not be possible with a well-fitting instrument. The test of a good fit, then, so far as the ear-tips are concerned, is that, first, they do not press too firmly upon the walls of the meatus, and, second, that it is not easy to understand conversation or hear other extraneous sounds without removing the instrument or directing its bell toward the source of sound. It is important that the ear-tips fit snugly, for on them rests the task of converting the meatus into a closed air-space, or, in other words, of preventing the vibrations of this air-space from dissipating themselves in the external air, or from becoming confused by the entrance of foreign vibrations. The external auditory meatus differs considerably in different persons with respect to the angle at which it penetrates the skull. It is essential therefore, that the ear-tubes shall be so curved as to cause the lumen of the ear-tips to coincide with the axis of the meatus. A stethoscope should always be selected with care, in order to make sure that these conditions are fulfilled. A pair of ear-tubes that fit perfectly will naturally be preserved with care by the possessor. It is perhaps superfluous to point out that, in order not to vary the conditions unnecessarily and to be sure of perfectly fitting ear-tubes, it is best in testing stethoscopes for their relative conduction of sound to use constantly one's own ear-tubes.

The bell of the binaural stethoscope was originally made of considerable size and of a flaring shape, with a view to cover the largest circle of skin that could conveniently be reached. This was done on the theory that the vibrations of the thoracic sound pass directly from the chest wall into the air within the bell, so that it is desirable to cover as large an area as possible. In stethoscopes of the Ford type the bell, of hard rubber or metal, or a combination of the two, has a conical shape with little flare, its diameter at the base being much less than that of many of the other types. This cone is solid, with the exception of a rather narrow canal that perforates its centre. In this type the conduction is almost entirely through the solid parts, so far as the bell is concerned, as is shown by the fact that sound is conducted about as well when the bell cavity

is plugged. But in this, as in all types of stethoscope, plugging the ear-tubes abolishes sound.

Whether a large or a small part of the vibrations is conducted by the walls of the soft rubber tubes, with which the bell is connected, depends upon their firmness and their thickness. Softness of material and thinness of walls are unfavorable for the conduction of sound, and especially of high-pitched sound; the more flexible the wall, the weaker and the deeper pitched the sound. According to Martini, the lumen of rubber tubes should be 5 to 7 mm., their walls should be 2 to 2.5 mm. thick, and their length should not exceed what is absolutely necessary—about 30 cm. (approximately one foot). In the judgment of the writer, 8 to 9 inches will suffice for the length. Such determinations of dimensions as those of Martini cannot be followed closely in practice, because of ignorance of the precise nature of the tubing that he studied.

The tubes should be of black gum rubber. Tubing of this material, which is sometimes strengthened and stiffened by an external layer of fabric incorporated with the gum, is obtainable in our markets. This is probably stiffer than the tubing tested by Martini (who distinguishes the grades tested as very soft, soft, and medium soft, and who obtained the best results with the hardest tubes and the medium soft), because he states that pinching the tubes between the fingers caused in all cases the complete disappearance of sound, which is not the experience of the writer with the type of tube last described. He was unable to prevent, by pinching, the transmission of the ticking of a watch auscultated through a stethoscope provided with such tubes. The stethoscope that the writer finds most satisfactory has a bell of the Ford type. The tubing used is of black rubber. The lumen of the tubes is 7 mm., the thickness of the walls 3.5 mm. The length of the tubes is 30 cm.

The soft rubber tubes of the stethoscope should not be bent during use if this can be avoided, for arching of the tubes impairs their vibratility.

We are now prepared to consider in a general way the conduction of sound through stethoscopes. All stethoscopes conduct sound from the chest wall both by their walls and by the contained column of air; but the relative amount of conduction by walls and by air differs widely in the various types of instrument. The more firmly any stethoscope is pressed against the chest wall (short of pressure so heavy as to prevent its vibration), the better the conduction. This is to be explained by more perfect closure of the air-space within the instrument, so that not only are the vibrations of the sound observed prevented from escape, but also extraneous sound is more perfectly excluded. Increased pressure

also gives better conduction through the walls of the instrument from closer contact with the chest. In the use of the monaural stethoscope pressure upon the chest, if exerted by the head of the observer, also causes more perfect coaptation of the surfaces about the ear with the top of the instrument. Pressure upon the tragus is prevented in the monaural stethoscope either by the prolongation of the central canal within a projecting earpiece which is inserted into the meatus, or, more usually, by hollowing out the upper portion of the cylinder so that the tragus may escape contact with the instrument.

It is, of course, important in auscultation of the lower-pitched sounds of respiration, and indeed of aërially conducted sounds in general, that the meatus be not occluded. Sound conducted by the walls of the stethoscope to the external ear is taken up by the walls of the cartilaginous portion of the meatus and passes freely thence into the air within. The union of the arms of the bell with the rubber tubes in the binaural stethoscope is never intimate and becomes weaker as the rubber deteriorates with age. Some sound is lost at this junction, and more from the small elastic force of rubber as a material. The vibrations of the rubber walls are transverse vibrations which pass easily to the air. How great a percentage of sound is lost in passage through the rubber depends upon its character and thickness. The greater part of the sound is usually, no doubt, carried by the air above the level of the bell. One thing is certain, which is that, once communicated to the air, these vibrations do not again return to solid media when the metal tubes of the earpieces are reached, but remain longitudinal aërial vibrations during the remainder of their way to the eardrum.

The joints between the ear-tubes and the rubber tubing are subject to the same evils as the joints between bell and tubing; but this is of less importance in their case, now that conduction has become largely aërial. Stethoscopes with a thin-walled flaring bell conduct sound mostly through the air; those with a heavy conical bell conduct a considerable portion of the sound by the walls, if the latter are of the proper thickness and firmness. Since there is this double conduction, no pains should be spared in developing to the utmost the sound-conducting properties of air-space and of walls. The subject deserves further study.

The relative amounts of air and of wall conduction in a given binaural stethoscope can be ascertained by plugging its bell. In some instruments this renders the stethoscope useless; in others it seems to make little difference; but plugging the earpieces abolishes sound conduction always. The story is told of one examiner, the bell of whose stethoscope was plugged with gum by his colleagues as a joke. He was quite unconscious of the fact, and went on tranquilly making diagnoses which his comrades were surprised to find were still as accurate as formerly.

One who is familiar with both types of binaural instruments will probably have learned that the sound heard through the stethoscope with a heavy bell is louder than that of the other type. Conduction is therefore better in the former. Neither type of instrument conducts very high-pitched tones well; but if there is any difference, it should be in favor of the stethoscopes which have good wall conduction, for high-pitched tones are better conducted by solids than by air. Fortunately, the great majority of tones produced within the chest are of low pitch, not exceeding, for the most part, 1000 vibrations per second as the maximum, and usually falling much lower than this maximum.

The unarmed ear is, however, still the best of stethoscopes, where it may be conveniently applied; and the student should not fail to practice immediate auscultation, and to resort to it frequently to check up his results, when there is any question of the presence of high-pitched tones.

Rubber tubing and conduction through solids may be profitably applied also in the analysis of breath-sounds. Thus, Müller says that, when he auscultated the tracheal breathing of healthy men through rubber tubes one to two metres long, he heard, to his surprise, pure deep vesicular breathing, and not a trace of the high tones that distinguish tracheal and bronchial breathing. But if he used a wooden rod, the high tones appeared distinctly.[10]

From what has been said it is evident that the results of auscultation will differ considerably according to the type of stethoscope used. Investigators who undertake the solution of problems in auscultation should ascertain the properties of their instruments, and in reporting their findings should state whether the stethoscope that they used conducts sound chiefly by the air or by its solid parts.

Having provided himself with a well-constructed and properly fitting stethoscope, the questions that the student should ask himself are: What do I hear through it, and what should I hear? Many who have never used a good and well-fitting stethoscope are ignorant of the possibilities of the instrument and, perhaps recalling early failures to make out sound that they expected to hear, are averse to exposing their supposed incapacity and avoid auscultatory diagnosis as much as possible. Others, with perhaps somewhat similar experience, are inclined to ascribe their shortcomings as auscultators to some slight impairment of hearing of one or both ears, which they may think permanently incapacitates them for the study of auscultation. Now, the fact is that many who have been indifferent to auscultation, when given a little instruction in such a way as to

[10] F. von Müller, *Verhandl. d. deutsch. Kong. f. innere Med. Wiesb.*, 1911, XXVIII, 184.

reveal to them their latent ability, take up the study with enthusiasm and success. And it is also a fact that slight impairment of hearing, a degree of deafness which is just noticeable in aërial conduction, is by no means an insuperable obstacle to the successful use of the stethoscope. For these reasons it is desirable that the inexperienced auscultator shall receive practical instruction in the use of the instrument; or that he shall at least measure himself with others and determine whether or not he can hear the sounds that they detect.

It is of great advantage in instruction that the teacher and the pupil shall hear identically the same sound. The instruments used purely for purposes of demonstration, in which several tubes are attached to the same bell, are not altogether satisfactory, for the reason that the rubber tubes are necessarily too long, and that the sound waves, being distributed among so many tubes, are so weakened that only rather loud sounds will reach all the ears. The same end can be more satisfactorily reached for one pupil by attaching the right tube of one and the left of another stethoscope to the same bell. The unused tubes being stopped, the instructor hears with his right ear the sound heard by the pupil with his left, or *vice versa*.' This method can also be used by the instructor for testing under the conditions of the stethoscope an ear, the hearing of which is known to be impaired.

Another method is to attach the right tube of one and the left tube of another stethoscope to one bell, and the remaining tubes to another bell. The instructor will then hear with his right ear the sound that reaches the left ear of the pupil from one bell, and with his left ear the sound from the other bell heard by the pupil with his right ear. The use of two bells is of advantage in comparing the sounds of two adjacent regions, and in following downward the wave of marginal sounds. (See p. 120.)

It having been ascertained in one of these ways that the pupil can hear as he should, the next question to be decided is, whether he can interpret correctly the sounds that he hears. The majority of pupils are easily capable of this after a little instruction. Occasionally one is encountered who seems psychically deaf, or who for some reason cannot, or will not, grasp the fine distinctions necessary to be observed. Instruction in such a case has been worth while if only to prevent the pupil from wasting further time on the subject.

In auscultation of the respiration the subject is instructed to breathe regularly and with as little noise as possible. It is usually best to demand a little deeper respiration than that of repose, and it is helpful if the subject can breathe mainly with his upper thorax while that part of the lung is under examination, and with the diaphragm and the lower

ribs when the lower part of the lung is being examined. To hear expiration distinctly, it may be necessary, in the case of persons with short and deep chests, to require them to breathe considerably more deeply than is their wont. Over the long and narrow chest expiration is usually heard without difficulty.

Normal breath-sounds are the sounds produced in the average quiet respiration of the healthy person. The loudness of the sounds depends upon the force of the respiratory movements and the thickness of the chest, their normal quality upon the proper position of the vocal cords. Normally, the sound of expiration as heard over the trachea is longer and louder than that of inspiration, because of the closer approximation of the vocal cords during expiration. The degree of separation of the cords at this time is not, however, fixed; the subject may diminish it to produce a sharper expiratory sound. This is particularly apt to occur when he is becoming fatigued in the course of a long examination. If the finer distinctions of sound are under investigation considerable care is necessary to avoid error from this cause. On the other hand, it is quite possible to open the larynx voluntarily so wide, that the sounds of inspiration and of expiration both disappear entirely. To determine the normal, that is to say the average, sounds of respiration, we must insist that respiration be neither abnormally noisy nor abnormally quiet; the vocal cords, in other words, must be in their usual position.

In the study of the breath-sounds, find first a place, usually over the lower lobe below the angle of the scapula, or in the lower axilla, where they appear to be normal. If there is any doubt as to their normal character, remove the stethoscope or direct the bell toward the mouth of the subject and listen to his respiration, correcting any faulty breathing. Normal breathing at the place of reference being secured, charge the patient to continue to breathe in the same manner, and compare frequently the breathing over the normal line and the place under examination, passing rapidly back and forth with the stethoscope between the two points, and checking up at once the way in which the subject is breathing, if there appears to be the slightest change at the point of reference.

THE BREATH-SOUNDS

The sounds of respiration, heard over the trachea or larynx, are harsh and relatively high pitched. Expiration is more prolonged than inspiration, its duration being to that of inspiration as five to four; and the sound of expiration is louder than the sound of inspiration. This is due to the

expiratory narrowing of the *rima glottidis,* which results in a greater velocity of the air-current and consequently in a greater production of sound, and which also demands more time for the escape of the inbreathed air than was required for its admission. The inspiratory sound heard over the lung is low pitched; it fills the entire period of inspiration. Expiratory sound is nearly inaudible over the lung during quiet breathing, and does not continue throughout the entire period of expiration.

All normal respiratory sound is caused by the air streaming through the rima glottidis. There are two kinds of respiratory sound, bronchial and vesicular, distinguished from one another by pitch and quality.

Bronchial sound consists of a group of relatively high-pitched tones, which are heard distinctly over the larynx and trachea, but are conducted but slightly through the parenchyma of the lung in expiration, and hardly at all in inspiration. Bronchial sound has the quality of an aspiration, as if the syllable *ha* were whispered.

Vesicular sound consists of a group of low-pitched tones which are heard best over the lung. This group of tones is present in both inspiration and expiration as heard over the trachea and larynx, but is difficult to detect there because of the loudness of the bronchial sound. The sound of inspiration as heard over the lung is made up almost entirely of the low-pitched vesicular sound. It is a soft "breezy" murmur, best imitated by shaping the lips to say *f,* and drawing in the breath.

Vesicular sound originates in the larynx from the vibrations of the vocal cords. This is proved by the fact that when, by the willed contraction of the laryngeal muscles, the cords are flattened against the sides of the larynx with an abnormally wide opening of the rima glottidis, this sound disappears both from inspiration and from expiration.[11] The sound that is heard over the lungs is fundamentally the laryngeal sound amplified by the thorax as a resonator. The appellation "vesicular" was given when it was supposed that the "vesicular murmur" originated in the air vesicles.

Bronchial sound likewise is produced in the larynx. It is amplified in the bronchi which act as resonators for sounds of higher pitch than that of vesicular sound. The names given to the two classes of respiratory sound do not indicate the place of origin, but the place of resonation of these sounds. That these sounds proceed from resonators is shown by the fact that the pitch of neither bronchial nor vesicular sound can be altered by increase or diminution in the force of respiration. (See p. 77.) Other tones may be added to the respiratory sounds when their force

[11] Bushnell, "The Mode of Production of the So-Called Vesicular Murmur of Respiration." *J. Am. M. Ass., Chicago,* 1921, LXXVII, 2104.

is increased, but the fundamental tones that characterize bronchial and vesicular sound remain unchanged as respects pitch.

The sound of expiration heard over the lung contains both the low-pitched tones of vesicular sound and the higher-pitched tones of bronchial sound. For this reason expiratory sound is sometimes called indeterminate breathing. The presence of bronchial sound as a characteristic of expiration heard over the lung is to be explained as follows. In order that the lung may best vibrate to the fundamental tone of the resonator in which it is contained, it must have a certain tension of expansion. That this is true is shown by the fact that quiet vesicular breathing is not heard equally well over all parts of the lung, but is heard best, or perhaps is heard only, over the part of the lung that is especially active at the time. The conditions for the production of the vesicular murmur are, therefore, most favorable when the lung is expanding in inspiration; and at the same time the conditions for the transmission of bronchial sound are least favorable, because the volume of the air-cells is greatest. On the other hand, in expiration the air-cells in their collapse permit the passage of a greater or less amount of bronchial sound, according to their volume at the place auscultated, and do not vibrate so well to the fundamental tone of the thorax. Since the mass of air-cells especially damps sounds of high pitch, the tones that pass through it from the bronchi will be the lower-pitched tones of the bronchial sound. Hence in expiration we have a combination of tones which is indeterminate; that is, it is lower pitched than the bronchial sound, but has not the characteristics of the vesicular murmur.

The breath-sounds, then, are divided into two groups: high-pitched sounds which are mostly modified by resonance in the air-tubes, and low-pitched sounds which are modified by the resonance of the lung within the thoracic cavity as a resonator. The first group will be called, for brevity, the bronchial component of respiratory sound, or, still more briefly, the bronchial component. The second group is the vesicular component. The sound of respiration as heard over the chest is taken as the norm. This sound contains a small proportion of the bronchial component, which is more distinct over parts in which the mass of interposed air-cells is relatively small; but this fact may be ignored in practice if the student is familiar with the normal variations in this respect. For the purposes of this discussion it will be sufficiently accurate to speak of the normal inspiration over all parts of the lung as identical with the vesicular component.

In health the vesicular component is conducted well through the chest, but the bronchial component, though really composed of relatively loud

tones, is almost entirely suppressed. The changes of conductivity of the lung in disease result almost always in an increase in the transmission of the bronchial component, with or without a marked diminution or a suppression of the vesicular component. That is, the normal vesicular breathing may be changed by the addition of higher-pitched tones, the change being so slight as to be detected only by the closest attention; or the higher-pitched tones of the bronchial component may overshadow the deep tones of vesicular breathing, so that the latter is not easily perceptible; or again, the vesicular component may have disappeared entirely, nothing being audible in the respiration except the high-pitched tones that proceed from the bronchi. To connect these changes of sound with the structural changes in the lungs that they indicate, we may say that the vesicular component is transmissible only through patent air-cells, and that the degree to which such transmission is impaired indicates the extent to which the air-cells have ceased to be patent, that is, have been filled with an abnormal solid or liquid substance, or have been compressed or destroyed. Or, to put the matter differently, the increased transmission to the ear of high-pitched sounds means that there has been an increase of some kind in the solid structures of the lung, as, for example, increase of fibrous tissue; or it means a solidification by exudate; or an obliteration by compression of the air-cells; or, finally it may mean the presence of a marked congestion of the blood-vessels or of an infiltration of the lung-tissues.

The bronchial component, if present to an abnormal degree, or found over regions in which it is not normally heard, is, therefore, an accompaniment of disease. The degree to which this component is increased gives a wonderfully delicate index as to the amount of impairment of the pulmonary tissues. It is important that one point should be brought out clearly, which is, what we mean exactly when we speak of high-pitched and low-pitched sounds. The sounds of breathing never consist of single tones; they are always combinations of different tones. Evidently, if such a combination contained, as it often does, tones of high pitch and of very low pitch, it might be called either high pitched or low pitched, according to the pitch of the tone to which particular attention is paid. Thus, the sound of normal expiration has been described by different authors as low pitched and as consisting mostly of sounds of medium pitch. Both of these statements are correct, but the authors who made them had different components of the expiratory sound in mind. The predominant characteristic of vesicular inspiration is the presence of the fundamental tone of the thorax, a tone of very low pitch. Add higher-pitched tones to the vesicular component, and the combination is usually

and properly said to be higher pitched than the normal, the emphasis being thus placed upon the newly added tones which reveal an abnormality. More commonly, the fundamental tone persists. Strictly speaking, the pitch of a sound is that of its lowest tone.

It is convenient to have a terminology which will indicate what the facts are with regard to the presence or absence of the vesicular component. Breath-sounds in which the bronchial component is increased, but the vesicular component still persists, are called bronchovesicular, the term implying the presence of both the bronchial and vesicular components. An attempt has been made to distinguish the mixtures of the two sounds in which the vesicular component predominates by calling them vesiculobronchial; but the distinction is little used, and does not seem to be of practical value. The important point to remember is that, however marked the bronchial tones, the vesicular murmur persists in some measure until the lung-tissues beneath the point of auscultation are completely solidified or otherwise deprived of air. The first evidence of disease is sometimes a weakening of the sound of inspiration on the affected side.

Addition of bronchial sound to the soft and low vesicular murmur imparts to it a sharper or harsher quality. Hence the expression a sharpened (*verschärft*) respiration, much used by the Germans, as a brief substitute for higher-pitched. The change is usually first noted and more marked in expiration but occurs also in inspiration. Authors recognize a sharpening of the respiratory sounds without prolongation of expiration, as, for example, in the normal breathing in the vicinity of the angle of the scapula; but, as a rule, sharpening of the sound is accompanied by a lengthening of the *audible* duration of expiration. The apparent prolongation of expiration is the more marked, the more distinctly bronchial the breath-sounds. It is hardly necessary to emphasize the fact that the prolonged expiration must not be conceived of as due to a real lengthening of the act locally from partial obstruction of the passage of air through the diseased parts, and the like; but as depending purely upon increased conduction of the normal sounds of the bronchi, the sound of expiration finally becoming audible through the entire duration of the expiratory phase, as the tissues grow more and more capable of transmitting bronchial sound with the advance of the morbid process; so that the ratio of length of expiration to length of inspiration over the lung comes to equal that obtained over the trachea. Only when fullest inspiration distends the focus of disease and there is a pause before the next inspiration, could there be a local lengthening of the duration of expiration; for it is inconceivable that expiration could be continuing in

one part of the lung if the remainder of it were beginning to expand in inspiration.

It is important to learn to recognize the slighter admixtures of bronchial sound in vesicular breathing. The attention should be concentrated closely upon all the characters of the sound—its pitch, loudness, timbre, and duration—and these characters should be borne in mind while the breathing over the symmetrical point on the other side, or other known normal part of the lung, is examined, repeating the process several times if doubt exists before a decision is made. It is here that diagnosticians most often err in their practice of auscultation.

The loud, harsh, and high-pitched breathing heard over the trachea or larynx is usually called bronchial breathing. Yet, as we have seen, there is here really an admixture of the low-pitched vesicular murmur. This may not be recognized by the examiner, who will nevertheless be struck by the difference in quality when he first hears the true unmixed bronchial sound. It would be more correct to say that bronchial breathing means the entire absence of the vesicular component, the bronchial component of respiratory sound alone remaining. Respiration of the purest bronchial quality is heard best over lung extensively compressed by tumor or aneurism, or completely solidified by pneumonic inflammation. The sharp distinctness of this bronchial sound *par excellence,* when once heard, will never be forgotten. It is comparatively rare in pulmonary tuberculosis, for the reason that extensive and complete compression or solidification of air-cells is not commonly a feature of the disease. If pure bronchial breathing is heard in a case supposed to be one of chronic pulmonary tuberculosis, the fact should be an incentive to inquire with care into the differential diagnosis.

Bronchial breathing as heard in pulmonary tuberculosis is, as a rule, of the type of tracheal breathing. The unmixed bronchial breathing is usually distinguished from this type by calling it pure bronchial breathing. It is illogical, of course, to define bronchial breathing as we have done, and yet to comprise within the classification kinds of breathing which retain somewhat of the vesicular component. Yet this use of the term is sanctioned by long usage; and if the distinction between bronchial and pure bronchial breathing is observed will not create confusion. As commonly used, the term bronchial breathing is to be understood to indicate breathing similar to that heard over the trachea, unless bronchial breathing *par excellence* is meant, in which case the adjective "pure" should be added. Bronchial breathing is also called tubular breathing. The French call it *souffle tubaire, souffle* meaning breathing or blowing. The term *souffle* is sometimes employed by writers in English.

As has already been pointed out, when the respiration is more vigorous than usual, the breath-sounds always have more of the bronchial quality; the bronchial sounds being louder, they are more perfectly conducted to the ear. In the breathing of children the small and very resonant thorax, as a resonator, has necessarily a higher pitch, so that sounds of medium pitch pass through it with greater ease than through the larger thorax of the adult. Possibly also the smaller masses of interposed air-cells permit a greater proportion of bronchial sound to reach the thoracic wall. For such reasons the breathing of children has a slightly bronchial or sharpened quality. Such breathing is called puerile breathing. The exaggerated breathing of the adult—as on the healthy side, when the other lung functions but little—is sometimes called puerile breathing. In this type of breathing the breath-sounds are slightly sharper than normal, and expiration is more distinctly heard and, therefore, may seem slightly prolonged. The distinction between puerile and pathological bronchovesicular breathing lies in the fact that, in the latter, expiration is harsher and distinctly longer. As a rule, however, the general dissemination over the lung of the exaggerated breathing prevents confusion with the breathing of disease.

Mixed breathing is that in which both vesicular and bronchial breathing are to be heard simultaneously, both types existing side by side, as it were, instead of being fused together as in bronchovesicular breathing. It is generally heard when the focus of disease is small, both vesicular and bronchial (or bronchovesicular) breathing being heard simultaneously. It is, of course, quite possible for normal and diseased tissues both to be present beneath the stethoscope bell at the same time.

Indeterminate, sometimes called indefinite (*unbestimmtes*) breathing, is simply a breathing in which the bronchial and vesicular components are both present, the respiratory sounds not being heard with distinctness, so that the precise character of the breathing remains doubtful.

Enfeebled breathing, in which the force alone is diminished, the vesicular murmur not being altered, is sometimes recognized over diseased areas, and by some authors is considered an important sign of early disease. It may be due to swelling of the mucous membrane of the bronchi or to the presence of secretion, and so be connected with an acute process; but its most common cause is pleural adhesion, which impairs the extensibility of the part. So far as it is due to this cause, the weakness of the breath-sounds may have no direct connection with the active disease, but may date back to conditions of many years' standing. Of course, bronchovesicular breathing may likewise be enfeebled for all of these causes. In view of the great frequency of pleural adhesions at the lung

apices, it is very unsafe to regard enfeebled breathing in this location as a sign of acute disease. Enfeeblement of breathing should of course be detected by comparison with the other lung. It is most satisfactorily determined when the respiration is not forcible.

In "cogwheel" or jerky breathing, the current of air is inspired intermittently. There is a succession of short puffs instead of a steadily flowing stream. In chronic pulmonary tuberculosis it is sometimes heard over healthy tissues adjacent to the lesion, as if there were some slight impediment to the admission of air, perhaps from a displacement of the bronchi as a result of the neighboring disease. As Turban puts it, cogwheel breathing presupposes rather large poorly functioning respiratory surfaces in the vicinity of infiltrations. By some authors it is counted an important sign in incipient tuberculosis, but it is also found in old and practically arrested processes. It is not, therefore, necessarily a sign of importance. From it should be distinguished "pseudo-cogwheel" breathing, which is heard over the entire lung, and is due to the inefficient or restrained action of the respiratory muscles, whether from the pain of pleurisy or from apprehension after hemorrhage, or, as sometimes occurs in examination, simply because the subject breathes carelessly. Occasionally the impact of the heartbeat when the heart is beating strongly causes the respiration of the adjoining lung to become jerky. The coincidence of the puffs with the rhythm of the heart betrays the nature of the condition.

Important to distinguish is rough, also known as impure or granular, breathing. In this type the breath-sounds are not clearly cut—hence the term "impure." The impression given is that of sound in tubes coated with moisture, and which is doubtless the condition present. "Impure breathing is a catarrhal symptom." Roughened breathing is, therefore, to be considered as a manifestation of the presence of moisture which, if the mucus were less adherent or in smaller tubes, would show itself as râles.

Cavernous breathing, or the breathing heard over a cavity, might be described as relatively low-pitched bronchial breathing with a cavernous quality. The cavity, generally of considerable size, has a relatively low fundamental tone and therefore reinforces the lowest of the bronchial tones. The cavernous quality is due to overtones, which are abundantly created if, as is usually the case in superficial cavities, the cavity has smooth and hard walls. Cavernous breathing, like the cavernous percussion-note, may have an amphoric quality. Sometimes the expression "amphoric echo" is used. In general, the pitch of the cavernous breathing is that proper to the volume of air contained within the cavity;

the quality depends upon the shape of the cavity and upon the smoothness and hardness of its walls. (See p. 105.) Persons who have a weak heart and are breathing more strongly than usual, as under examination, are apt to accentuate and prolong the expiratory sounds, because expiration in their case is to a considerable extent an active muscular act, the accessory muscles of expiration being called into play. Hence expiratory sound becomes more marked in their breathing and, among expiratory sounds, especially the fundamental tone of the cavity. For this reason probably the statement is sometimes made that the pitch of expiration in amphoric and cavernous breathing is lower than that of inspiration. But, so far as the fundamental note of the cavity is concerned, this is of course impossible.

The walls of deep-seated cavities are not usually smooth and hard. The overtones characteristic of cavernous and amphoric breathing are therefore absent. The breathing is low pitched; inspiration is almost inaudible, and expiration much prolonged and notably low pitched. The prolongation of expiration is probably to be explained in the same way as in the case of the superficial cavity. As for the lowness of pitch, since sounds, which are able to penetrate the patent air-cells at all must be low-pitched sounds, whatever the vibrations produced in the cavity, evidently we can hear only the sound waves that result in tones of the lowest pitch, especially the lowest tone, the fundamental tone of the cavity. The breath-sounds of a large cavity resemble vesicular breathing for two reasons: first, in the absence of the overtones which characterize the typical cavernous sounds; second, because the lowest tones alone are transmitted to the ear.

Metamorphosing breathing is a sound beginning with a sharp hissing and passing into a soft bronchial breathing. It is said to depend upon the sudden enlargement of the lumen of a partially obstructed bronchus.

PATHOLOGICAL CONDITIONS WHICH MODIFY BREATH-SOUNDS

Changes that Affect the Entire Lung.—Any morbid condition of the respiratory apparatus which influences the rate and the force of the respiration, such as dyspnea due to inflammatory swelling or to spasm, changes the character of the breath-sounds. The stridor of the partially obstructed larynx, the dyspnea of asthma and even the increased force of the breathing of acute bronchitis modify the respiratory sounds throughout the lung. The changes in such conditions are such as would be ex-

pected in breathing which is louder than usual; the bronchial component of the breathing is more marked over the chest because it is actually *ab origine* a louder sound. The character of the breathing as it is heard over the chest in asthma is also modified by obstruction or contraction of bronchioles that involves so much of the lung, as to necessitate extra effort to force air through narrow tubes partially filled with a tough mucus. Hence there arises a variety of local modifications of the breath-sounds, wheezing, sibilant and musical sounds. In this group of morbid conditions the breath-sounds are changed for the whole lung at the larynx, and are further modified by the addition of adventitious sounds due to the presence of secretion in the smaller air-tubes.

In general the pitch of the breath-sounds is raised in disease, the conditions usually favoring an increase of the bronchial component. An exception to this rule is found in emphysema. In this disease the transmission of bronchial sound is diminished, the volume of the air-cells being increased above the normal and thus interposing a greater obstacle to the passage of high-pitched sounds. The pitch of the breath-sounds is therefore lower because of loss of the bronchial component. It is lower also so far as the fundamental tone is concerned because the size of the thoracic cavity is increased and because the lung-tissue has a slower rate of vibration due to the relaxation of the lung.

Localized Pathological Changes in Their Effect upon the Breath-Sounds.—In diseases in which only a portion of the lung is affected, such as pneumonia and tuberculosis, in the absence of dyspnea or other cause for noisy laryngeal breathing, deviations from the normal sounds of respiration are to be heard only over the diseased parts. Alterations in the sounds heard over these parts are due to a change in the conductivity of the tissues for sound which results, broadly speaking, always in increase in the transmission of the bronchial component, and in decrease in or absence of the vesicular component. The following conditions are instrumental in effecting this twofold modification of respiratory sound:

1.—Fibrous tissue arising from indurative inflammatory processes replaces or compresses the air-cells, thickens locally the peribronchial and perivascular fibrous coats and, reaching the surface of the lung, transmits sound from the bronchi, the force of which has been less diminished by passing through air-cells than is normally the case.

2.—The atelectases which result from the implication of bronchus or bronchiole in a localized tuberculous or other inflammatory process partially collapse the tributary air-cells and thus diminish the transmission of the vesicular, and increase that of the bronchial component.

3.—Solidification from inflammatory infiltration or from caseation permits sound to pass from the bronchi which is normally suppressed by the air-cells, conducting it through what has become a homogeneous and solid acoustic medium to the surface of the lung, or, in deeper lesions, diminishing the breadth of the tract of air-bearing tissue to be traversed. At each change of direction of the bronchial tubes, one portion of the sound is reflected onward within the tubes, another portion passes through the walls, this portion being larger the greater the curvature and thinner the wall. Sound cannot be driven through tubes of less than 1 mm. diameter. Below this diameter conduction can take place only through the air-cells. Ultimately, therefore, in health sound passes from the bronchi to the surrounding parenchyma there to be for the most part suppressed by the air-cells. But if the lung is solidified the bronchial sounds are transmitted to the ear with little loss. The walls of the normally flexible small bronchi when stiffened by inflammatory infiltration also transmit more perfectly the vibrations communicated to them by continuity from the walls of the larger tubes, thus practically bringing the vibratile portion of the bronchi nearer to the surface of the lung. Infiltrations which surround the bronchi are supposed by some authors to increase their vibratility.

4.—Compression from tumor, cyst, abscess, or aneurism within or adjoining the lung, or from pleural or pericardial effusion, drives out the air from the parts affected, thus suppressing the vesicular component, and by solidifying the lung favors the transmission of the bronchial component.

It is often assumed that the altered breathing heard over a lesion instead of being due to a change of conduction of normal sound is abnormal sound produced locally. Citations might be made, which would show that in the opinion of some authors there is reason to suppose that air is driven with locally increased force through tubes narrowed by inflammatory swelling, or by the presence of secretion or neighboring tubercle, and even that locally increased respiratory function must be assumed to be the cause of sharpened vesicular breathing in the neighborhood of old tuberculous processes without catarrhal symptoms. Also that the lengthening of the expiratory sound in advancing early tuberculosis is due to progressive diminution of the calibre of the bronchi in the affected part of the lung which leads to "a localized expiratory dyspnea."

It will at once occur to the reader that since neither vesicular nor bronchial breathing is changed in pitch by increase in force of the air-current, we cannot invoke a more forcible flow of the respired air locally

as a cause for the change from vesicular to bronchovesicular breathing. But even granting sound could be thus changed, is there any means by which the force of the breathing could be increased locally? Unquestionably the thorax expands and contracts as a whole under the stimulus of the central nervous system. The stimulus of the two halves of the thorax being equal, the response of the muscles of the two sides will be equal, except so far as it may be altered to the prejudice of the affected lung by spastic conditions of the muscles of that side of the chest, and the expansion of the two sides will be proportionate to their relative expansibility.

There can be no local increase of function in parenchymatous lesions; for, well-marked local lesions have lost their elasticity; they neither expand nor contract during the respiratory movements to any considerable extent. So long as the lesion persists the respiratory capacity of the lung is lowered by the amount of lung-tissue that does not functionate. There being no obstacle to the expansion of the healthy side, the air flows freely into the lung, but with quiet breathing the diseased side has comparatively little exchange of air, the slight pull of the thorax being met by the prompt expansion of the healthy side, but being for the most part ineffective on the diseased side. With deeper breathing the relative inelasticity of the diseased lung expresses itself visibly by a delay in expansion, the thorax rises more slowly on the affected side. The healthy lung expands quickly at first but toward the end of inspiration, being already well filled with air, its expansion is comparatively slight. On the diseased side the continuing pull of the thorax overcomes to a greater or less extent the resistance opposed by the inelasticity of the lung; it too is forced to expand to the degree permitted by its inelasticity, but its maximum dilatation occurs toward the end of the inspiratory act. This delay in expansion is known as lagging. It is simply evidence of relative inexpansibility of the diseased lung. This inexpansibility is due to many causes, acute infiltrations, temporary plugging of bronchi with mucus, spastic conditions of the muscles on the affected side and the adhesions, semi-atelectases and fibrosis of old tuberculosis. Pleural adhesions and fibrous processes are the commonest causes in chronic pulmonary tuberculosis. Some authors claim that lagging is evidence of an active inflammation and state that the distinction between lagging and relative inexpansibility is that in lagging the inspiratory expansion is delayed but becomes complete, while in the latter the expansion of the lung is defective at the end of inspiration. But it is generally admitted that lagging is a permanent sign of massive fibrosis in healed, or healing tuberculous processes

and that the significance of the sign must be determined by further study of the case. A lagging of the diseased side with final complete expansion can only be conceived of in case of spasm of bronchi or muscles.

Air admitted to the lung in ordinary quiet respiration does not force its way through partially obstructed tubes, at least not equally and contemporaneously with its entrance into healthy parts of the lung; it goes to the places to which it can go most easily—to the diseased lung up to the limit of expansion which is possible under the conditions of elasticity and the force of the breathing and no farther, however slight may be the result in actual increase in size, and also in greater volume to the healthy lung. Adjustment of the loss of respiratory surface is always bilateral. In so far as the diseased lung has become inelastic its expansibility has diminished. Though no tissue may have been actually destroyed, as a physiologically active organ it is diminished in its efficiency. The other lung in so far as it is compelled to functionate more actively is correspondingly increased. This increase in volume is attended by a lower position of the diaphragm and a pushing over to the diseased side of the mediastinum and the heart.

Inelasticity of the diseased lung shows itself also in a loss of mobility of the diaphragm which is unable to draw down the lung as freely as it does in health. This is sometimes apparent in even early and comparatively slight tuberculous lesions. The phenomenon, first described by Williams, has received many and complicated explanations, but in the writer's judgment it may be very simply accounted for in the majority of cases as being the visible expression of the inelasticity of an even slightly diseased lung.

The effect in general of the pull of the chest wall upon the position of the thoracic organs when the lung is diseased is a subject that will be considered in another connection.[12] It will be necessary, however, to anticipate that presentation of the subject to some extent in order to take up the relations of the so-called compensatory emphysema to the question of localized provision for increase of respiratory function. When a region of the lung is involved in an inflammatory process it becomes stiffened, at first by congestion and infiltration, later, by fibrotic contraction, so that it loses much of its elasticity and can no longer follow the pull of the thorax. The rise of the chest in inspiration tends to create a vacuum which should be normally filled by the air entering the lung beneath. In so far as a focus of disease does not expand, either the movements of the thorax are restricted, or air is supplied from elsewhere to fill the vacuum. This need is met for the most part by the expansion of

[12] See: Displacement of the Heart in Disease of the Lung, p 124.

the other lung, and probably adequately met when the respirations are quiet. But the lung is not perfectly elastic and the compensation on the part of the other lung is afforded in an indirect way. Hence deep breathing, and especially the full inspirations of a coughing fit, cause an especial temporary traction over the unexpanded region of the lung. This is answered by dilatation of the surrounding healthy lung which is called compensatory emphysema. This dilatation is furthered also by the positive pressure during cough. In general, compensatory emphysema is of more or less complete disappearance from the percussion-note of the evino especial clinical or diagnostic importance, except as explaining the dences of a small old localized lesion. The significance of compensatory emphysema in the present connection is that it affords evidence that the lesion does not expand in inspiration and that air is not driven through the lesion with increased force. Such effect as a lesion may have in increasing locally the force of the respiration seems on the contrary to be expended upon the neighboring healthy parts of the lung. Another proof that the increase of the bronchial component of the breath-sounds in disease is not caused by local changes in the acoustic properties of the air-tubes is afforded by the experimental widening of the rima glottidis. In a case in which the subject was able to open the glottis more widely than normal, bronchovesicular breathing which had been audible over an obsolete tuberculous lesion of the lung disappeared when the larynx was fully opened.

In general, then, it may be said that the character of the breath-sounds heard over a focus of disease is not changed materially by their passage through the air-tubes of the lesion. The sounds are primarily the same sounds as those of health, as a rule. Such changes of sound as may be noted over a lesion are due to differences in conduction of these normal sounds.

The foregoing being the general rule as to the production of sound within the bronchi, it remains to note briefly some exceptions.

Considerable force of the air-current is necessary for the production of sound in the continuity of a smooth-walled tube, especially when, as in the bronchial tree, the individual tubes are short and relatively wide. Experiment shows that the force of normal breathing is too slight to create sounds anywhere except at the vocal cords, but that sounds are elicited from the larynx by a very weak current of air. Here we have something resembling a musical apparatus—the cords have been likened to the reeds of musical instruments. Now, reeds function by intermittently breaking the current of air. To produce sound in the bronchi with the force of the air-current that normally passes through them, all that is required is

that some condition exist capable of creating vibrations by rhythmically interrupting the current of air.

The air may be interrupted in the following ways: A bronchus may contain a coating of mucus, not lying in semi-inspissated clumps, but thin enough to coat smoothly and evenly the walls of the tube. This mucus, if ruffled by an air-current, is thrown into waves which may act as valves to interrupt the passage of the air to a certain extent. The result is rough breathing.

The mucus may be more ropy and lie in detached masses which under cough or specially strong breathing may be driven forward in such a way as to temporarily fill the tube and, passing on, to leave behind them threads of mucus hanging in the lumen which vibrate and produce musical sound —musical râles.

Or, the mass of mucus may be forced through the tube by the air behind it, the air finally bursting through it and producing moist râles.

The air may pass from a small bronchus to a larger one and bubble up through a pool of mucus, producing bubbling or gurgling râles.

And, lastly, sound may be produced by localized stenoses of the bronchi. Sound is created much more readily in a tube that is narrowed at one or more places than in a tube of uniform calibre, the constriction breaking the smooth flow of air. Tumors of the bronchi of course afford many possibilities of valve action, but stenosis, usually of a limited and clinically unimportant extent, is most commonly due to tuberculosis. It is well known that the projection into the smaller bronchi of an inflammatory swelling due to a tubercle which has pierced the bronchial wall is not uncommon. Such processes run their course generally in small tubes, and if they do not heal lead quickly to collapse of the tributary air-cells, so that the conditions are most unfavorable for the free flow of air which is necessary for the production of stenotic sounds. What air may still be present below the stenosis in such lesions may properly be assumed to be stagnant, as a rule, on account of the inelasticity of the affected tissues. However, the possibility of the exceptional production of stenotic sounds in this and similar ways must be admitted.

RÂLES

Râles are sounds produced in tubes or other air-spaces of the lungs by the movement of air in the presence of liquid, or in some cases in the separation of moist surfaces by the entrance of air. According to their

quality of sound râles are classified as dry or moist. Dry râles may be crackling (or crepitating) or "sticky" or sibilant. Moist râles may have the quality of sound described by the terms, serous, mucous, viscid, bubbling, gurgling, sonorous, musical, metallic, amphoric. As respects size, râles may be fine, small, medium-sized, large or coarse.

When air is forced through a tube plunged into a liquid, it escapes in bubbles the size of which will depend upon the calibre of the tube, and which leave the orifice of the tube, as Talma [13] shows, in the form of a hemisphere. At first this hemisphere bends upward until it comes to resemble a ball of air, connected by a tail with the air of the tube, then suddenly breaks loose and rises as a sphere to the surface. Here assuming again, when isolated, a hemispherical shape, it bursts in a longer or shorter time, according to the viscosity of the liquid. The gurgling or bubbling sound which is familiar when air is blown through a liquid is not caused by the bursting of bubbles upon the surface; if a sound is indeed caused by the bursting of the bubble, it is extremely faint and of quite a different quality. The gurgling sound which precedes in point of time the bursting of the bubble is produced according to Talma, by the vibrations excited when the liquid, which has been displaced during the protrusion of the bubble from the tube, springs back to occupy its former place as the bubble detaches itself.

When two "tacky" surfaces, that is, surfaces which are moistened by a liquid of some viscosity, are suddenly torn apart, a sound is produced which, the viscosity remaining the same, varies in intensity and in quality according to the rapidity of separation of the surfaces. When the separation is very slow there is no sound; with increasing velocity the sound is at first faint and "moist" in quality, but soon grows "drier" until with high velocity it becomes a hard, dry crackle, a series of miniature explosions which do not give the impression of being connected at all with liquid, resembling more the crackling of fire in brush. As Geigel well points out, even with water the moist quality of sound is only produced when the movements that displace the water are slow, as for example, the sound of dripping or of the gentle stirring of water. When water is expelled from a hose-pipe, if the stopcock is but partially open and the flow of water is relatively slow, the sounds will be liquid in character. But when water mingled with air leaves a nozzle under high pressure the sounds are hard and crackling. The same is true of the impact of water; the sound will have less of moist quality the greater the force with which

[13] Talma, "Beiträge zur Theorie des Rasselns," *Deutsches Arch. f. klin. Med., Leipz.*, 1876, XVIII, 53.

the water strikes against a hard surface. Similarly if the hands are rubbed gently and slowly together the sound is soft and low pitched, but a brisk rubbing produces a high-pitched and dry sound.

The degree of "dryness" in the sound depends likewise upon the viscosity of the liquid. The force of separation of the moist surfaces remaining the same, a very viscid liquid gives dry sounds; with lessening viscosity the sounds become more and more moist in quality and fainter until they finally disappear altogether.

While a bubble necessarily implies the presence of air, the separation of more or less agglutinated surfaces and even friction within joints or between folds of fascia may produce sounds very similar to those of bubbles, though no air can be present. This is shown to be true by the adventitious sounds of the thorax outside of the lung proper which are discussed in Chapter III (see p. 112).

Stones struck together under water produce vibrations of an intensity that is painful to the ear of the diver as every boy knows. But a dry twig snapped under water gives rise to a very similar sound. This illustrates the fact that a sudden displacement of matter may cause vibrations communicable to the ear as sound, independently of the presence of air.

Adventitious sounds within the lung may be produced first, by the bursting of bubbles, second, by the detachment of bubbles from submerged orifices, third, by the separation of tacky surfaces. The first causes the ordinary moist râles; the second, gurgling or bubbling râles; the third, crepitant râles.

With regard to moist râles the statement that they are due to the bursting of bubbles is founded rather upon authority than upon knowledge. So far as the ear can decide, the sound is certainly the bursting of a bubble. There is the effect of a miniature explosion, a "pop" with a suggestion of a delay as if an obstacle were overcome as the preliminary to a rupture, this delay being more marked the more viscid the fluid. Whether small volumes of air forced through a tube together with liquid might not produce a louder sound when they escape from the liquid than bubbles, which rise by their buoyancy and burst on the quiet surface of a liquid, is a question which the experiments of Talma do not decide.

From what has been said as to the physics of the production of râles it is evident that the term "dry" râle is a misnomer. There is no distinct line of demarcation between the moist and the dry râle; as the liquid becomes more viscid the quality of the râle becomes drier. Since the force of respiration does not change materially, the quality of the râle thus affords an indication of the degree of viscosity of the liquid present, which

furnishes important information in the appreciation of the case. One cannot study too carefully the sound of the râles heard. Among several observers who make the distinction between dry and moist râles there will be found to be no agreement as to what constitutes dry or moist râles—there is a range of what might be called medium viscosity in which opinions differ so widely that the terms become meaningless. But their use is so well established that it would not be wise to attempt a change in the terminology. A certain class of sounds which make up a considerable proportion of what are called dry râles are not râles at all, but extrapulmonary sounds of some kind. It is to be remembered that the râle formed in a markedly viscid liquid should have a characteristically delayed and well-marked bursting effect. A distinctly audible and not very distant "dry" sound which is atypical in this respect is probably not a pulmonary râle.

Râles of the ordinary kind, being produced in the presence of mucus, may be called mucous râles. The mucous râle is characterized by qualities of sound that denote the presence of a liquid of more or less viscidity. The distinction between the mucous râle of high viscidity and the sound of separation of tacky surfaces on the one hand, and muscle and fascia sounds on the other, is not always to be made from the quality of the sound alone. Of course the term "râle" is not properly applied to any sound which is produced independently of moisture.

Varying from the mucous râle in the opposite way from the dry râle is the so-called "serous" râle, the least viscid of all râles. These are heard rarely in the chronic lesions of pulmonary tuberculosis under conditions which are little understood. The effect is as if the lesion had been flooded with a non-viscid fluid through which air bubbles. The râles are very numerous, burst without the characteristic delay of the mucous râle, as if the rupture of the bubble were not attended with resistance, and have a peculiar soft breezy quality which cannot be described. The phenomenon is generally evanescent and is not inconsistent with a considerable prolongation of life, or even with the arrest of the tuberculous process.

The French word "râle" was originally applied to tracheal râles, the death rattle and other sounds audible to the bystander. The gurgling or bubbling râle is produced in large air-tubes or cavities in the presence of abundant fluid, and when it is not a sign of impending death or of profound anesthesia is most often heard in advanced cases of tuberculosis. Being produced, as a rule, in large tubes in the presence of varying amounts of more or less tough mucus, the sounds vary greatly in pitch and quality. Since they depend upon accidental and changeable rela-

tions of mucous masses to tube orifices, the sounds are evanescent and are easily caused to disappear or to become changed by cough or deep breathing. The vibrations of the coarser gurgles are so strong that they may be readily detected by palpation.

The rhonchus is properly the coarse bubbling or rattling râle of the large bronchi or of the trachea. The term is sometimes used as equivalent to râle in general. There is no object in thus introducing a synonym into the nomenclature. The term rhonchus as defined above has the advantage of indicating with one word a class of râles which otherwise would require several adjectives for their description.

The râle is usually conceived of as an intermittent sound. The sound of rough breathing is due, as we have seen, to the presence of mucus upon the walls of the bronchi, which impedes the progress of air and produces a slight change in the quality of the breath-sounds. If, however, the fluid within the tube is of greater viscosity and the air moves with considerable force, the fluid may be ruffled into waves which act as valves or reeds (such as those of the organ) which by intermittently breaking the flow of air may excite vibrations in it. The tubes then behave like organ pipes and emit tones peculiar to their length. Hence the so-called musical râles. There is a series of more or less musical sounds, ranging from the coarse gurgling râles of low pitch to which the resonance of the bronchi imparts a sonority—the sounds being plainly enough due to the detachment of submerged bubbles—to the sounds described by the words growling, snoring, rattling and the like, in which the intermittency of the sound is apparent and, the pitch rising, to the more purely musical sounds from the tonal vibrations of the bronchi in which our ears detect no intermittency and finally to the high faint whistle of sibilance, a sound with little if any resonance. The Germans distinguish musical sounds of this nature from râles. They speak of growling, snoring, whining and hissing, not of growling or snoring râles, etc. The distinction is a logical one if intermittency, as appreciated by our ears, is the distinguishing feature of the râle. The importance of the râle however does not lie in its character as a sound, but in the information that it furnishes as to the kind of fluid present in the air-passages and its location. If there are coarse gurgling râles there is abundant not very viscid mucus in the largest tubes; if the sounds are higher pitched and more musical the smaller bronchi are concerned, the mucus is more viscid and more scanty, and so forth; the information thus obtained being of the highest value in the estimation of the nature and location of the morbid process.

Râles differ essentially from breath-sounds as to their place of origin.

The breath-sounds originate in the larynx and find in the masses of air-cells and in the bronchi resonators, which alter more or less their original character. On the other hand, râles originate in the various air-spaces of the respiratory apparatus below the larynx, their character being, of course, usually more or less modified by the air-chamber in which they are formed. This is true whether the sounds are caused by the bursting of bubbles, by bubbles detached from submerged orifices, by the valve action of tenacious mucus or by the separation of sticky surfaces. Some name is needed for the group of sounds which originate elsewhere than in the larynx and the usage in English is to employ the word râle independently of the intermittency of the sounds thus designated. Therefore while the distinction above referred to is new, the fact that it can now be made seems an additional reason for continuing to extend the application of the word "râle" to include continuous sounds which originate in the deeper air-passages. The râle, then, may be defined as a sound which is produced in the displacement by the respiratory air of fluid contained in the air-passages.

The crepitant râle, an exceedingly fine crackle close to the ear and best imitated by the crackling of salt in the fire, or the rubbing of hair between the fingers held near the ear, is best explained as the separation of minute surfaces covered with a viscid liquid, whether air-cells or respiratory bronchioles is immaterial. The râles are always of the same size, occur in showers and only during inspiration.

Some German writers use the term "crepitation" indifferently with the term "râle"; more commonly they use the word crepitation to denote the crackling râle. Neither practice is to be recommended, for crepitation is a word of wide applicability, so that when used as a substitute for the word râle, or for a certain kind of râles, there is often doubt as to the precise sound which it is intended to describe.

The term "subcrepitant râle" implies a relation to the crepitant râle. The important relation is that of succession in the same pathological process. The subcrepitant râle, a very small moist râle, occurring in showers in inspiration only, follows the crepitant râle in the evolution of pneumonia. Its smallness should be insisted upon as a marked characteristic. Yet it is larger than the crepitant râle, therefore formed in a different place. Its moist quality shows the presence of a liquid of less viscosity and, therefore, probably of greater abundance than that associated with the crepitant râle. The term "subcrepitant," if not applied strictly to the râle of the pneumonic process, is superfluous for it would simply denote a small moist râle. It should, therefore, be restricted to the small moist râle of pneumonia.

The writer desires to emphasize the importance of accurately distinguishing crepitant and subcrepitant râles from other small râles. This distinction which is almost universally neglected even by systematic writers, enables the diagnosis of pneumonia to be made with accuracy, even though the pneumonic patch be small, and is of special value in the diagnosis of tuberculous pneumonia. The distinction is made by the size of the râle—crepitant and subcrepitant râles are smaller than other similar râles that appear in showers. The student should not fail to take advantage of every opportunity to study cases of undoubted pneumonia and should impress upon his mind with much care the size of the râles heard.

We speak of a given râle as large and moist, small and viscid and the like, unless we intend to designate the râles belonging to the pneumonic process when we use the terms crepitant and subcrepitant. The size of the râle shows sufficiently, as a rule, the size of the bronchus in which it is formed. If the râle belongs to a cavity it would naturally be designated accordingly.

Râles are also classified as consonating and non-consonating râles. In the former the sound has high overtones that give it a more or less musical quality. "Consonating" properly means: "sounding together with another sounding body." This is true of the consonating râle only in the sense that it is generally produced in a resonator, a normal or abnormal air-chamber of some kind with firm walls which permit the formation of high-pitched overtones. The consonating quality of sound is not carried through the air-cells of the normal lung. The fact that it is present is, therefore, usually proof of either solidification of the lung or the existence of a superficial cavity. The writer knows a physician, who can produce moist râles somewhere in his air-passages heard in expiration followed by cough, when the bell of the stethoscope is held before the open mouth. These râles are consonating râles. The consonating quality is also often present in the sounds of loose cough. This quality which is producible in normal bronchus and larynx is therefore, not necessarily evidence of a serious pathological condition. Such evidence is only afforded by abnormally good thoracic transmission of high-pitched tones. It is probable that nearly all râles are originally consonating. As Martini says: "bronchial sounds are robbed of their higher partial tones in their passage through air-containing lung and thus acquire a 'washed out', non-consonating character." [14] The non-consonating râle is defined negatively—all râles that do not have the consonating quality are non-consonating.

[14] Paul Martini, *Deutsches Arch. f. klin. Med., Leipz.*, 1922, CXXXIX, 257.

Amphoric and metallic râles are heard under the same conditions as those that produce amphoric and metallic breathing.

Morbid Sounds that Simulate Râles

Sounds arising from morbid processes in the thoracic wall which simulate râles and friction-sounds should be briefly considered. Coplin suggests that pseudopleural sounds may result from the myositis which sometimes accompanies pleurisy and might produce audible friction from the movements of septal fibres or fibre sheaths and bundles, and states that Rosenbach considers the sound of new leather a muscle phenomenon.[15] It is well known that in advanced cases of tuberculosis inflammatory processes may extend beyond the parietal pleura with the production of a burrowing suppuration, which may even result in a fistula discharging externally. Processes of this kind might easily lead to the simulation of râles, the discrimination of which from true râles is, however, of little practical importance under the circumstances.

Sewall [16] has reported a case of pneumothorax which showed strong "new leather" sounds over the whole area to the left of the sternum from the first intercostal space to the sixth and out to the anterior axillary line. On respiratory motion there was marked tactile fremitus which seemed superficial. The new leather sounds changed their character generally so that they came to resemble a dry pleural friction-rub. At autopsy the left lung was completely collapsed and the cavity of the pleura contained no fluid. Here it would seem probable that the sounds were due to spasm of the intercostal muscles.

Friction of inflamed surfaces in the upper abdomen may result in sounds heard at the base of the lung which simulate râles. A patient once came to the writer with the diagnosis of tuberculous pleurisy, who had jaundice, high fever, profuse sweats, an enormously enlarged and very sensitive liver and atrocious pains shooting to the right shoulder. Auscultation near the base of the lower lobe of the right lung anteriorly showed indistinct and intermittent sounds which resembled distant râles more than friction-sounds, and were evidently propagated from the dome of the liver. The patient made a good recovery and has remained in excellent health. His lungs were perfectly sound.

Pleural Friction Sounds.—The sounds caused by the rubbing together of the surfaces of the pleura when roughened by inflammatory

[15] Coplin, *Proc. Path. Soc., Phila.*, March, 1904.
[16] Sewall, *Am. J. M. Sc., Phila.*, 1909, CLXXIII, 364.

exudate or by miliary tubercle are called pleural friction-sounds. They may be harsh or soft, evanescent or of long duration. They are most frequently heard over the lower lobes where the gliding movement of the leaves of the pleura is most marked. The character of the sounds usually suffices to indicate their source, but occasionally sounds of the nature of gurgling râles simulate closely the "sound of new leather" or other creaking or rubbing sounds that might be referred to the pleura. Friction-sounds are also said to imitate râles; in fact one writer has gone so far as to attempt to prove that all of the finer râles are really pleural friction-sounds. Theoretically, the separation of tacky surfaces of the pleura might result in sounds identical with that of the more or less viscid râle and the distinction in the given case may be impossible. Marginal sounds which imitate râles to perfection are undoubtedly due to such a cause. And this, as we have seen, is the usual explanation of the genesis of the crepitant râle. There is, then, no absolute distinction to be made by the ear between râles in general and friction-sounds. Anatomically, they are distinguished by their place of production; râles are produced in the lung; friction-sounds in the pleura.

VOCAL RESONANCE

Vocal Resonance of the Normal Chest.—The voice may be controlled as to loudness and to some extent as to pitch. Its place of production is known. In so far the auscultation of the voice seems a simpler problem than that of the breath-sounds. But it is really much more difficult on account of the complexity introduced by the varying amount of reinforcement given to the more or less pure musical tones of the voice of widely varying pitch.

Auscultation over the lungs of one who speaks results in hearing everywhere the voice tones reproduced indistinctly and confusedly, the sound is a humming sound. The words that are uttered are entirely unintelligible. The voice is somewhat louder over parts of the lung in which the mass of air-cells is thin and large bronchi are near, as in the upper part of the upper lobes anteriorly and especially near the sternum, between the scapulae behind and, sometimes, immediately beneath the clavicles, especially the left clavicle. It is only when the conditions favor reinforcement by the resonation of the thorax that the transmitted sounds are loud over the deeper portions of the chest. Just as a tuning fork is best heard when its pitch is that of the fundamental lung-note, so the voice is best transmitted through that part of the chest which is adapted by its size to

resound to the tone sounded. Accordingly the heavy bass voice is well transmitted through the lower chest; the voice of women is better carried through the upper chest, but rarely with the same relative intensity as the bass voice.

Bronchophony, Whispered Bronchophony.—The increased transmission of the voice through the lung in disease is called bronchophony. Objection has been made to this term on the ground that there is a normal bronchophony—the voice is carried through the chest to some extent in health. If we define bronchophony as the increased transmission of the voice through a diseased part by virtue of the improved conduction of sound which arises from the presence of solidifications and indurations, we have a single word which contrasts the mode of transmission of sound in disease with that in health in which reinforcement of low-pitched and obstruction to the passage of high-pitched sounds are the predominating characteristics. Variations in the transmission of the voice through the normal chest depend upon the varying relations of tones of different pitch to the resonator through which they are transmitted. For a tone of a given pitch and timbre, therefore, the degree to which it will be transmitted is a permanent characteristic of the transmitter, the thorax and its multilocular contents. On the other hand the conduction of sound through a diseased region of the lung is dependent upon the condition of the thoracic walls and of the lung as they are affected temporarily or permanently by the disease. The presence of pleural adhesions and, still more, of a liquid exudate checks to a greater or lesser extent the vibrations of the thorax both locally over the focus of disease and as a whole. Adhesions persisting from an earlier pleuritis may also permanently impair the vibratility of the thoracic wall, and diminish its conduction of sound. The effect of disease, therefore, is often to impair the conductivity of sounds which depend upon the vibrations of the resonator as a whole, i. e., low-pitched sounds. But the disease so far as it affects the lung-parenchyma usually increases the conductivity of the part. To the degree that the lung has become solidified or indurated it is a heterogeneous sound medium. It does not vibrate in unison with the remainder of the lung. If the lesion consists in many small pneumonic foci the lung-tissue will be relaxed between the foci and the result will be a tympanitic note upon percussion and a somewhat increased transmission of the voice, because relaxed lung is relatively airless lung. Fibrous indurations, or infiltrations, or pneumonic consolidations, or combinations of any or all of these conditions diminish or remove entirely the air-content and increase the solid parts of the lung. The affected portion of the lung then has become, or tends in a greater or less degree to

become, a single homogeneous sound medium through which sounds are conducted with little loss of intensity. And if the lesion is fused to the chest wall by fibrous adhesions the loss of sound by reflection at the costal pleura is done away with and the conduction of sound is still farther improved. While as we have seen the effect of the lesion has usually been to cripple the vibratility of the thoracic walls both locally and generally, it is the low-pitched sounds which are especially affected. Sounds of high and of medium pitch are but little, or not at all, influenced. Hence voice-sounds formerly suppressed or muffled by the air-cells are now conducted with ease through the solid acoustic medium.

In rare cases vocal resonance has been found to be transmitted very imperfectly through fibrosis. This is probably usually to be accounted for by the presence of exudate, plastic or liquid, in the pleural cavity. Deformation of the lung from contraction of fibrous tissue with resulting partial collapse and obstruction or kinking of bronchi may possibly play a rôle here. In the case of the most massive solidifications of pneumonia the lung is sometimes so increased in size that it is furrowed deeply by the ribs and actual bulging of the intercostal spaces may be present. The effect of the pressure thus shown to exist is sometimes to annul the vibrations of the thorax on the diseased side and thus to abolish the reinforcement of sound from that source. The pressure also damps the localized vibrations of the diseased lung so that voice-sounds are very poorly transmitted through it.

But, as a rule, the voice-sounds are conducted with considerable force through the solidified lung of pneumonia, as well as through the indurated lesions of an old pulmonary tuberculosis. In these cases the airlessness of the lung or the presence of fibrous pathways which connect bronchus with chest wall, permit sound to pass from the bronchus without the damping effect exercised by normal alveoli. Higher-pitched sounds may now pass through the lung. In marked bronchophony at times only very high-pitched sounds may be heard.

The pitch of the voice-sounds is a more essential element in the diagnosis of solidification of the lung and of cavity than their intensity. Elevation of pitch upon the side suspected of disease, as compared with that of the healthy side, is proof of the absence or diminution of the restraining effect exercised by air-bearing lung upon the transmission of the higher-pitched sounds. The absence of voice-sounds and of breath-sounds is sometimes due to special conditions of sound conduction in the bronchi. A plugging, temporary or permanent, of the bronchus which passes to the region auscultated suppresses transmitted sound. In gangrene vocal resonance sometimes persists, but in the writer's experience

is more frequently lost, in which case the bronchus is probably either obstructed by collapse or filled with fluid. A marked diminution in sound conduction is sometimes to be ascribed to the filling of the bronchus to a greater or less extent with froth.

The Whispered Voice.—Some persons are not good whisperers; they either whisper inaudibly or if they seek for loudness, they phonate. This is because they have not learned to approximate the vocal cords properly. A whisper is produced by the partial closure of the glottis as is shown by the fact that the sound ceases at once if the glottis is opened wide. The sound of the whisper and the breath-sounds are produced by the same mechanism. The transmission of whisper through the chest wall is, therefore, governed in general by the same laws as those which regulate the transmission of the breath-sounds.

Both voice and whisper are normally conducted through the portions of the lungs in which the bronchi are near the surface, in the upper part of the upper lobes and especially in the vicinity of the bifurcation of the trachea on the right side in front. (See p. 111.) The precise extent of the conduction of whisper, which is naturally not so well carried as the voice, varies greatly in different persons according to the conformation of the thorax, so that it is useless to attempt to indicate the normal limits. These are usually obtainable in the individual case by auscultation of the normal side of the thorax. It might be expected that the sounds of whisper would be heard whenever breath-sounds to which they are so closely allied are audible. Why should not, for example, the mere thread of fibrous tissue which sometimes conducts the bronchial element of bronchovesicular breathing so remarkably, conduct also a whispered pectoriloquy? This it practically never does. In general the transmission of whispered sounds is uncertain. They constitute a valuable sign when present, but there seems to be a certain capriciousness in their appearance in disease which interferes with their usefulness in diagnosis to a marked degree.

Weak and high-pitched sounds, while not capable of reinforcement to the same degree as the tones of the voice or those of musical instruments, nevertheless undergo a certain amplification when produced in or near a resonance box. (See p. 78.) The sound of a scratch within the piano case is enormously louder than that produced by a similar scratch upon a board in the free air. A whisper is produced by a much closer approximation of the vocal cords than that which takes place in unrestrained expiration. This approximation having been effected by a willed contraction of the laryngeal muscles, the vocal cords have a definite tension and the sounds produced, though due to the vibration of the

edges only of the cords, have a definite pitch and are thus in some respects analogous to the more musical and louder tones of phonation. The sounds of whisper, from its mode of production, will usually be of much higher pitch than the respiratory sounds produced by a larger and more slowly moving current of air and are, therefore, less capable of exciting the resonance of the thorax.

The foregoing remarks apply especially to whisper produced, as it usually is, without thought of pitch. A little experimentation will show that it is possible to make very high-pitched whispered sounds by almost closing the glottis, elevating the larynx and directing the current of exhaled air upward. On the other hand, a deep whisper may be produced which must nearly approximate the pitch of respiratory sound and presumably, therefore, experiences more or less reinforcement by thoracic resonance. Practically, however, whisper is usually relatively high pitched and like other high-pitched sounds is suppressed by passage through normal or nearly normal lung, even though under the conditions present bronchovesicular breathing may be audible. On the other hand Sahli remarks that bronchophony, especially for whisper, may be heard over a consolidated area although bronchial breathing is indistinct, a phenomenon which is probably to be accounted for by the difference of pitch of the two series of sounds. In general the transmission of sound through solidified lung depends upon the pitch of the sound under conditions the nature of which we can only conjecture. Much of the apparent capriciousness of whisper is thus to be explained.

We may expect therefore a certain dissociation between vocal and whispered bronchophony and still more between vocal and whispered pectoriloquy. It is hardly to be expected that the conditions of sound conduction through solidified lung should permit both vocal and whispered sounds to be heard in the same degree of perfection, at the same time and in the same place in a given case. In superficial cavities with resonant wall, however, sound is amplified through a wide range of pitch and both vocal and whispered sounds may be often heard with distinctness.

Pectoriloquy, Whispered Pectoriloquy.—The voice in health is transmitted through the chest wall as a confused sound. Hence we speak of vocal resonance rather than of voice transmission. The tones may be more or less distinct; the words uttered are indistinguishable as such. This is chiefly due to the fact that consonants are transmitted with difficulty.

Vowels are distinguished by the relative intensity of their partial tones. Every vowel has one or two tones that are characteristic of it, the "formatives," the absolute pitch of which is constant. These tones depend

upon the shape of the mouth cavity which is especially adapted to them individually. According to Helmholtz,[17] these characteristic tones are (continental pronunciation throughout): for U, f (170 vibrations per second); O, b′ (460); A, b″ (910); E, f′ and b‴ (341 and 1815); I, f and d⁗ (170 and 2280). Words containing the vowels, I, E and A have a high-pitched character, those with O and U a deep character. Some of the consonants also possess characteristic tones which lie mostly in the three-lined octave (1000–2000). These are most marked in consonants which can be prolonged, such as S, Z and "sh." Words that contain many vowels and consonants with high-pitched formatives are especially deformed in passing through the healthy chest; on the other hand; words with but few high partial tones, such as "hundred," are transmitted with relatively little change. The conditions of sound transmission in the diseased chest are, therefore, the more indicative of advanced pathological changes the more perfectly words of the former type are enunciated. "Chaise" and "seashells" are suggested as appropriate examples; "acht und sechszig" is used in Germany.

If the region of the lung which separates the bronchus from the chest wall is completely solidified, the consonantal sounds are well conducted and the words spoken are sometimes transmitted with startling distinctness. The voice then seems to proceed from beneath the part auscultated —hence the name "pectoriloquy," or "speaking in the chest." Pectoriloquy is due to the same causes and is produced in general under the same conditions as pure bronchial breathing. It furnishes important evidence either of cavity or of complete solidification of some kind. The same remarks apply to whispered pectoriloquy which denotes the perfect conduction of whispered words.

Egophony.—The term egophony, or "goat-voice," is given to a form of bronchophony in which the voice has a peculiar nasal quality and a tremulousness which causes it to be likened to the bleating of a goat. It is most frequently heard just above the level of a pleural effusion and has no special pathological significance. Montgomery finds reasons from experiment for ascribing it to conditions in the pathways of sound which permit a special reinforcement of the nasal tones of the voice. The tremulous quality has been supposed to be due to the vibration of a thin sheet of liquid interposed between the ear and the source of sound. The alternate expansion and collapse of air-cells and of bronchioles, which must occur at the upper surface of the pleural exudate as its level rises

[17] Helmholtz, "Lehre von den Tonempfindungen," 1913, 168 ff. Quoted by Martini, *Deutsches Arch. f. klin. Med., Leipz.*, CXXXIX, 257. See also, Helmholtz, *Popular Scientific Lectures*, 1895.

and falls with the varying intrathoracic pressure during speech, might, perhaps, best account for the tremulous character of the voice sounds. Egophony, however, sometimes occurs when in all probability there is no effusion.

Vocal Resonance in Pleural Effusion.—When a liquid exudate collects within the pleural cavity the lung-tissue retracts by virtue of its elasticity and becomes correspondingly relaxed. This is shown in the increase of vocal resonance often found above the level of the exudate. But other factors act to diminish the transmission of sound. These are:

1. The introduction of a heterogeneous sound medium. In the healthy chest the transmission of sound is practically from air to chest wall. In the case of effusion the liquid constitutes a sound medium which is very different from air; there is a considerable loss by reflection as the sound enters the liquid on its way to the chest wall. There is also some loss by diffusion, the greater the larger the quantity of the liquid.

2. Interference with the resonance of the affected side. The presence of a liquid within the cavity interferes with the action of the resonator. The part of the thorax below the level of the exudate is checked in its vibrations and reinforcement is correspondingly diminished, the effect being the same as when water is introduced into a thin-walled tumbler or musical glass. In this case the pitch of the tone produced by striking the glass is raised in proportion to the amount of water present, that is, the part of the glass below the level of the water no longer vibrates to produce an audible musical note.

The collapse of the smaller bronchi coincidently with the relaxation of the lung is probably compensated for by the increased conductivity of sound due to the relative airlessness. But if the exudate continues to increase, especially if it increases to the extent that the elasticity of the lung has become exhausted and actual compression of lung-tissue occurs, the larger bronchi in part collapse. Less sound, therefore, enters the affected lung, for Montgomery has shown that of two tubes connected with a glass Y the shorter transmits the less sound. The fact that the still patent bronchi of the compressed lung are farther removed from the ear is probably not of much importance here, water being an excellent conductor of sound.

Usually, then, the transmission of the voice is impaired or almost abolished in the presence of a pleural exudate because of impairment of the vibratility of the thorax and because of the loss of sound by reflection and diffusion and, in the more marked cases, by compression of the bronchi, these factors more than compensating for increase in the transmissibility of sound as the result of the partial relaxation of the lung. The

complete suppression of vocal resonance by pleural exudate is, however, very rare.

But not very rarely cases are seen in which the vocal resonance is not impaired but, on the contrary, is of normal or even greater loudness notwithstanding the existence of an exudate in the pleural cavity. Hitherto in our discussion it has been assumed that the lung is normal except for changes directly due to the pleurisy and that the gain in the transmissibility of sound from the relaxation of the lung is more than offset by the loss from reflection, etc. But if we suppose that the lung, instead of being simply relaxed, is solidified, we account for the maintenance of a resonance practically equal in force to the normal because not only does the solidified lung conduct sound much better than the relaxed lung but there is also much less reflection of sound on its passage from solid tissue to water than there would be if it passed from air to water. Another explanation for the preservation of normal vocal resonance in cases of pleural effusion is that adhesions from a previous pleurisy bind the lung so firmly to the chest wall at the place auscultated that it cannot contract. In other words, so far as conduction through the part connected by adhesions is concerned, there is no pleural exudate to interfere with the transmission of sound. And not only may there be no interference with vocal resonance but the resonance may actually be increased, because adhesions firm enough to resist the pressure of the exudate are frequently associated with fibrous changes in the underlying lung, this being especially common in pulmonary tuberculosis; and this fibrosis may conduct sounds much more strongly than the normal lung. If on the other hand there is no fibrous change of moment in the underlying lung, the adhesions being relics simply of a past pleurisy, the effect of the adhesions would be to diminish the vocal resonance since they would interfere with the vibrations of the part of the chest wall that is implicated.

Voice Transmission in Cavity and Pneumothorax.—Large pulmonary cavities as a rule involve the superficial portions of the lung. Their external wall contains little air-bearing tissue. Under such conditions the sounds of the voice pass with little diminution to the ear; they are generally louder than the sound heard through the normal chest. When the external wall of the cavity is formed practically by the thoracic parietes and the pleura, as is frequently the case in a large cavity in portions of its extent, the conditions favor resonance and the sound may be much increased, occasionally to an intensity with strong voices which is actually painful to the ear. The conditions favor also the transmission of whisper. Pectoriloquy, vocal and whispered, is common in cavity when the external wall contains no air-bearing tissue.

But even a thin layer of patent air-cells diminishes the transmission of the voice to a remarkable degree. In the case of deep-seated cavities the walls generally consist of air-bearing tissue through which only low-pitched tones are transmissible. Pectoriloquy is, therefore, absent and the voice-sounds may not be characteristic of the condition. Diminution of vocal resonance which is infrequently observed over the more superficial cavities may be due to the interposition of air-bearing tissues or to the filling or partial filling of the cavity with liquid. Sometimes however the phenomenon is difficult of explanation.

In pneumothorax vocal resonance is as a rule diminished and more markedly diminished than would be the case with an equally large liquid exudate. Sometimes voice-sounds are entirely absent. When a pulmonary fistula affords a direct communication of the bronchus with the air in the pleural cavity, or when the bronchus is separated only by the wall of a thin-walled cavity, the vocal resonance may be increased—the pneumothorax then is practically equivalent to a cavity from the acoustic standpoint. It is readily apparent that a bronchus, imbedded in airless tissue which extends to the surface of the lung, if still patent, might likewise transmit the voice-sounds through the pneumothorax with considerable intensity. This explanation seems the most probable one for cases which have been reported in which there was no direct aërial communication and no closely bordering cavity, yet the vocal resonance was increased. This phenomenon, when it has been observed in artificial pneumothorax, has been ascribed to peculiarly favorable conditions of tension of the contained air. But, manifestly, in the class of cases for which artificial pneumothorax is especially indicated, improved conduction from solidifications within the lung is to be expected. According to Fishberg whispered pectoriloquy is sometimes exquisitely marked in localized pneumothorax.

VOCAL FREMITUS

Vocal Fremitus in Health.—The chest as a resonator vibrates to the sound of the voice. If its vibrations are strong enough to be clearly perceptible to touch the phenomenon is known as fremitus. Vocal fremitus is the more marked the more nearly the tone sounded approaches the fundamental note of the thorax. For this reason fremitus is best obtained in health over the lower lobes of the lung of the adult male. Fremitus is occasionally to be made out over the upper chest of women who have strong and clear voices. The right lung is larger than the left

lung; the right main bronchus is larger than the left and is more nearly vertical. Anteriorly the trachea is in more direct contact with the chest wall of the right side. For these reasons, probably, fremitus is as a rule rather more marked upon the right than upon the left side of the thorax. It is customary to palpate for fremitus while the subject counts aloud. Words beginning with consonants the enunciation of which breaks the current of expired air at or near the lips, "n" and "th," and the labials are best adapted for the explosive utterance which causes the thorax to "hum" most strongly. "Ninety-nine" is a favorite vocable. The dependence of fremitus upon the thorax as a resonator is shown by the fact that it is best elicited when the subject produces the lowest clear tone of which his voice is capable.

In practising palpation for fremitus it is better to apply the flat palm, or, preferably, the ulnar edge of the hand, than to use the finger tips, except for the discrimination of the degree of fremitus in small areas, when it is imperative the finger tips be used. Symmetrical points of the two sides of the chest should always be compared. Some examiners use both hands at once, bilateral palpation, but if the differences sought for are minimal it is advisable to apply the same hand successively to one and the other side. The hand should be pressed firmly to the chest wall and, in the comparison of the two sides of the thorax, it is important that the pressure should be equal. Fremitus may sometimes be elicited more distinctly in the male patient if he speaks in a deeper tone than that which he generally uses in conversation, or better still if he sings with a deep bass voice.

Vocal Fremitus in Disease.—Fremitus is increased or diminished in the same way in general as resonance in the conditions of disease. The changes produced by disease in the general resonance of the thorax have even a more marked effect upon fremitus than upon vocal resonance. Fremitus is usually much increased in solidifications and indurations, as for example in hepatized pneumonia and in the fibrous lesions of an old pulmonary tuberculosis. It is abolished in considerable effusions of liquid in the pleural cavity. As in the case of vocal resonance, vocal fremitus may be preserved, notwithstanding the presence of a moderate amount of exudate, if a solid lung lies behind the exudate or if strong adhesions hold portions of the lung in apposition with the chest wall. Conditions which favor the increased conduction of vocal resonance likewise result in the increase of fremitus. The intensification of sound being no longer solely due to the general vibrations of the thorax, but now depending upon local conduction of sounds which are forced upon the diseased lung from the bronchi, fremitus is produced by sounds of

higher pitch. The vibrations to which the fremitus is due are no longer generally diffused over the thoracic wall but are strictly localized to the area which overlies the focus. In testing for fremitus the difference between the vibration of the two hemithoraces is of course the important point. This difference is naturally the more marked the higher the pitch of the sound that produces fremitus on the diseased side. We should, therefore, call for high rather than for low tones and women should be tested as regularly as men, for a woman's voice which is quite incapable of causing the normal chest to vibrate may give well-marked fremitus over an infiltrated or indurated pulmonary area.

The permanent obstruction of a bronchus abolishes fremitus over the region of the lung which it supplies; the plugging of a bronchus with mucus may lead to the temporary loss of fremitus which reappears when the plug of mucus is expelled by cough. The absence of fremitus over a lesion is of course only of significance if it is present over the healthy lung. Occasionally fremitus may be entirely absent over the lower lobe of the affected lung in chronic pulmonary tuberculosis, the transmission of vocal resonance remaining fairly good. In this case some condition is usually present which impairs the vibratility of the thorax while at the same time there is insufficient increase of sound conduction to produce fremitus locally. An example would be a large tuberculous lesion of a lower lobe, consisting of numerous as yet discrete foci and without pneumonic infiltration, which is overlaid by a dense and thick plastic pleural exudate connecting the whole or the greater part of the affected lung so intimately with the chest wall that the vibrations of both lung and thorax are greatly impeded. Here the exudate abolishes the well-marked vibrations necessary for the general reinforcement of sound and hence for the production of general fremitus, but at the same time the lung-tissues are not sufficiently solidified to force localized vibrations strongly upon the chest wall. This phenomenon has been called by Kuthy the dissociation of fremitus and resonance.

In the foregoing the endeavor of the writer has been to set forth the more common and easily explained facts of vocal resonance and vocal fremitus. While exceptions have been referred to, no attempt has been made to state, certainly not to account for all the rarer phenomena, the pathological curiosities, of physical diagnosis, some of which are difficult of explanation. Voices of every pitch, of various timbres and of all degrees of strength, lungs with every conceivable variety of pathological densities, recent or long standing, thoraces of various shapes and sizes and of widely varying natural resonance which is often strongly influenced by pleural adhesions and pleural exudate all lead to widely vary-

ing possibilities of reinforcement and conduction. When we reflect that the complicated acoustic phenomena of voice conduction are observed to a large extent with the aid of stethoscopes which themselves are far from perfect in their transmission of sound and that their peculiar excellence in some, their serious defects in other, respects are often not considered by those who use them, or at all events are not reported by them, we shall not find it strange that difficulties are experienced in harmonizing and in accounting for all of the observations that are of record. Because of such difficulties of interpretation, because vocal resonance and vocal fremitus at times seem to fail or to mislead, it is often remarked that these signs are unreliable and that they rightly have less vogue than was formerly the case. But we may be sure that, however uncertain the results of our observations may seem to be, the transmission of sound always takes place according to fixed laws. It is for us to ascertain what these laws are. The complexity of the problems presented to us is one of the elements of their fascination.

CHAPTER III

PHYSICS AND PHYSIOLOGY IN RELATION TO THE PHYSICAL DIAGNOSIS OF DISEASES OF THE LUNGS

SOUND

Sound is due to the vibrations of an elastic body which are communicated to the ear through the air or otherwise.

A tone is a sound of a single and regular rate of vibration. A noise may be the sound of a single disturbance of the air which, not continuing, has no periodicity. But unless a noise is practically instantaneous it is a collection of discordant tones which, under favorable conditions, may be analyzed so that the pitch of the individual tones that compose it may be ascertained.

A clang is a tone with its harmonics. What we commonly speak of as tones are really clangs, that is, they are accompanied by overtones. The timbre, or quality of a sound is determined by the nature and number of the accompanying overtones.

Sound produced unconfined, as in an open field, is propagated in spherical waves in all directions until an obstacle is reached, when the sound waves are reflected. In sound, as in light and heat, the angle of incidence of the wave is equal to the angle of reflection. If the body upon which the sound impinges is capable of penetration by the sound, the extent to which the sound will penetrate the body will depend upon the angle at which the sound waves strike it.

Sounds passing through a medium suffer a loss due to absorption—a certain portion of the energy of the sound waves is converted into heat and motion, this portion being the greater the softer and the less elastic the medium concerned. There is also a considerable loss of sound through reflection at the surface of each of the media through which it may pass.

Sound upon entering a medium travels in all directions through its mass. So far as respects the intensity of the sound in its propagation in any given direction, there is considerable loss from this cause which is called diffusion.

The intensity of sound is inversely as the square of the distance of the

sounding body from the ear. This law, however, does not apply to sound carried in tubes, if they are straight, not too wide and have smooth walls. In such tubes the sound waves, being reflected by the walls of the tube, are forced to proceed in the direction of the axis of the tube and may be transmitted far with little loss. The speaking tube is a familiar example of this.

The vibrations of sound are also carried far in an elastic solid medium, if they originate in that medium. This is true even of extremely faint sounds. Tap gently with the head of a pin upon the edge of an iron bathtub and apply the ear to the tub. The sound though it may have been inaudible through the air is then distinctly heard. And it is heard as well if the ear is applied to the most remote part of the tub—that is, at the point on the other side of the tub opposite the point struck. The faint sound in this case causes vibration of the entire mass of the tub, but is too feeble to pass from a solid to the air without total loss. Similarly, the scratch of a pin upon a long steel bar is heard with ease by the ear applied at the other end of the bar, though the free ear at that distance hears nothing. The whole mass of the bar has evidently vibrated as the result of the disturbance of a few of its particles by the scratch. Holding the same bar lightly, pass a small metallic rod gently along its surface in its long diameter. If the bar is of good steel and is not in too close contact with other solid bodies, very pleasing musical sounds are produced. In this case also vibrations have been excited throughout the mass of the bar, but with results very different from that of the pin-scratch. In the latter case longitudinal vibrations have been caused by a force which has supplied sufficient energy—and well-applied energy—so that the mass of the bar can vibrate in the way that is characteristic of its size and elasticity. But why is a musical tone, or tones, produced in the one case and not in the other? To answer this question we must first consider what it is that makes a musical tone.

A long cord is fastened to a wall. The other end of the cord being grasped in the hand, the cord is made to undulate rapidly and regularly. The waves of the undulation proceed to the fixed end of the cord and are there reflected back towards the hand. But, the undulations continuing to be produced, those that travel towards the wall meet those reflected from it. In some parts of the cord the summits of the advancing and reflected waves will coincide, in others the summit of one wave coincides with the trough of the other. In the first case the vibrations will be increased in amplitude; in the second they are neutralized. The cord will thus arrange itself into a series of vibrating loops that are visible to the eye. The parts between the loops in which there is no undulatory move-

ment are called nodes. Experiment has shown that a musical tone is produced in the same way by the much more rapid vibrations of the violin string. The sound waves passing to and fro, the advancing waves meet the returning waves and produce invisible loops and nodes in the same way as do the undulations of the cord. The longitudinal vibrations of the steel bar which we have supposed, running rapidly to and fro through the mass of the bar, act in a very similar way to the vibrations of the cord and of the violin string, and also form the loops and nodes that are characteristic of musical sound. Such vibrations peculiar to the vibrating mass are called free vibrations. The experiments with the bar show, therefore, that an elastic body to which vibrations capable of producing sound are imparted responds in two ways. First, it simply vibrates throughout its mass, or as far as the energy of the vibrations permit. If the energy of the vibrations is sufficient, the vibrations may be communicated to other sound media with which the first body is in contact, but the vibrations simply pass through the medium, or media, experiencing no change, except a gradual loss of their intensity, until they finally cease. Such vibrations are called progressive sound waves. Second, the vibrations, if not drawn off into other bodies in contact with the first, that is to say, if they are not damped, have more than sufficient energy than simply to traverse the mass. They run to and fro within it and thus inevitably interfere with one another and arrange themselves so as to produce the tone characteristic of the size and elasticity of the given mass. Sound waves of this kind are called stationary sound waves because the vibrating loops and the nodes remain stationary as respects their location in the string or other vibrating body through the duration of the individual sound. In order to result in musical sound the sound waves must be longer than the mass in which they originate, so that they can be, as it were, folded upon one another, which is another way of saying that they must undergo reflection before musical sound is produced. When such musical sounds reach our ears they come as progressive sound waves due to the vibrations which the stationary sound waves of the instrument have communicated to the surrounding air. Sound moves through the air, not in transverse waves like those of the musically sounding violin string, but in longitudinal waves of alternate condensation and rarefaction. Sound waves advancing free through the air progress indefinitely without alteration of their character until they finally die out. But sound waves in confined air may experience reflection and thus produce the nodes and loops of musical sound. Any volume of air, confined within more or less vibratile walls, to which suitable vibrations are imparted will form stationary sound waves and will emit a sound which, except under

special conditions which will be considered farther on, is characteristic of its volume. By tapping a wooden box we determine by the sound whether the box is large or small, empty, partly full, or completely full of some solid substance. That is, we expect the law to be invariable that the series of vibrations excited in the box will not be the same as those caused by the tapping (which in itself would be the same for all wooden boxes of the same kind and thickness of wood) but will depend upon the natural rate of each box, that is, chiefly upon the volume of air that is within it. A large empty and closed box will give forth a deep tone, a smaller box a higher-pitched sound. Increase in the force of the blow produces a louder sound but the pitch of the sound is not raised. We may conclude from these experiments that every volume of confined air under atmospheric pressure which is surrounded by vibratile walls has its own peculiar rate of vibration which is constant so long as its shape and size remain unaltered.

In Fig. 14, A denotes a row of particles at rest. If they are disturbed by the transmission of a transverse wave, they will be displaced, as

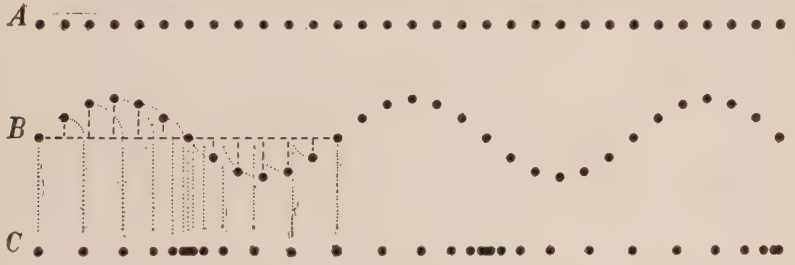


FIG. 14.—Transverse and longitudinal displacements. A, a row of particles at rest. B, transverse displacement of the same particles. C, longitudinal displacement. (Miller, "The Science of Musical Sounds," The Macmillan Company.)

shown in B, forming a curve. Assuming the displacements of the particles to occur in a vertical plane, the curve resembles that of waves of water. The dotted line in B would represent a horizontal line away from which the transverse displacements take place and, in the case of water waves, would represent the surface of still water. The maximum displacement of the particles upward is equivalent to the crest of a wave of water, or the top of the loop of a vibrating cord. The maximum displacement downward is like the trough of the wave. The crest of the wave and the bottom of the trough are the places of greatest motion; after the analogy of the undulating cord they are called loops. The points at which the horizontal line cuts the wave are places of no motion and are called nodes (or knots, as if the vibrating cord were motionless at these points because actually tied fast to the horizontal line.

A wave is measured from crest to crest. The horizontal line drawn in B shows the first wave as beginning in a node at the point B and extending to the first node of the second wave. There are two points at which the line cuts the curve in each wave, therefore two nodes; and there are two loops, one of maximum displacement upward, the other of maximum displacement downward. The wave is therefore divided into four equal parts, each part being the distance from a loop to a node, or from a node to a loop.

If the displacements were longitudinal instead of transverse vibrations forward displacements along a horizontal line would take the place of upward displacements and vice versa, and the wave would be a longitudinal wave with alternations of condensation and rarefaction, that is, with pressure changes. The sound waves of air have longitudinal vibrations. Longitudinal displacement of particles is shown in Diagram C, Fig. 14. Fig. 15 represents at B longitudinal waves which may be compared with

Fig. 15.—A, transverse waves. B, waves of compression. (Miller, "The Science of Musical Sounds," The Macmillan Company.)

tranverse waves of the same length shown at A. The points at which the lines are crowded together in B represent nodes of condensation, the widest spaces midway between two nodes of condensation represent nodes of rarefaction. The motion of particles (shown as lines in B, Fig. 15) is greatest midway between a node of condensation and a node of rarefaction, that is, at the loop, and is always towards a node of condensation and away from a node of rarefaction. There is no motion at the nodes. If a ruler is laid horizontally across the page at A in Fig. 15, it will be noted that the nodes of condensation and rarefaction shown in B correspond to the points at which the ruler cuts the wave of A—that is, they correspond with the nodes of B, Fig. 14—and that the crests of the waves in A correspond with the position of points in B which are midway between nodes—that is, with the loops of B. The fine lines drawn from B to C in Fig. 14 connect the individual particles and show

their movements in the two kinds of displacement. These diagrams show that longitudinal waves may be conveniently represented by the diagram used for transverse waves and this is commonly done.

There are two kinds of organ pipes, flue pipes and reed pipes. In the flue pipe a strong current of air is forced through a very narrow slit and is directed against a sharp edge, known as the lip, after which it partly enters the pipe, partly passes into the external air through the mouth of the pipe. The incoming air is caused to vibrate in this manner with many and various vibrations, but nothing approaching a musical note is produced, for if the pipe fails to "speak" only a loud hissing noise is heard. The upper end of the flue pipe is usually open. The pipe being open at both ends, the ends are necessarily loops, for at these points the air of the pipe is in contact with the outside air; in the production of the fundamental tone the node is situated at the centre of the pipe. Since the wave-length is always four times the distance between a loop and the nearest node, it follows that the wave-length is twice the length of the pipe, the vibrations traversing the pipe twice before all of the phases of a single wave have been passed through. If the pipe is closed at both ends (which is only done in experiments) the ends are necessarily nodes and the loop is in the centre of the pipe. Here also the wave-length is double the length of the pipe.

The sound produced in a closed box, especially if one of its dimensions is much greater than the others, is analogous to the doubly closed pipe. In this case the nodes are situated at the extremities of the longest dimension, and the wave-length will be twice that dimension. In a box of approximately equal dimensions the pitch will be governed by the volume of air within the box. In the pipe closed at the top, but like the open pipe, open at the mouth, the node will be at the top, the loop at the bottom of the pipe; the wave-length will therefore be four times the length of the pipe and the pulse must traverse the pipe four times before the wave is complete. The open organ pipe does not always respond with its fundamental tone. By properly increasing the force of the air the harmonics of the primary tone can be brought out successively, the fundamental tone being weak or absent. In this case the air is divided into additional nodes and loops.

In the organ pipe used as a musical instrument it is always the fundamental tone of the pipe that is desired and the force of the blast is therefore adjusted in order to bring out that tone. The flue pipe is tuned only at the pipe. If the tone is too low the column of air may be shortened by the insertion of a partition near the top of the pipe; this is sometimes done. But since it is not possible in practice to lengthen a pipe

when a lower tone is wanted, the most convenient way of tuning, when the deviations from the normal tone are slight, is to alter the width of the upper orifice of the pipe by bending more or less a projecting flexible plate, the principle being, the smaller the orifice the lower the tone. These facts show that if the column of air is of proper length and shape there is no difficulty as to the formation of vibrations that will elicit the fundamental tone.

All organ pipes are open at the bottom. Those closed at the top are spoken of as closed pipes. The reed pipe is usually a closed pipe. In these pipes a flexible and elastic plate of metal called a reed is so placed as to vibrate in the incoming air-current. By this means the rapidity of the vibrations that are to call forth the desired tone can be regulated. The closed pipe also can be compelled to sound the harmonics of the fundamental tone by increasing the force of the blast, but in this case a different series of harmonics is produced because the arrangement of nodes and loops is different from that of the open pipe. The reed pipe must be tuned at the reed as well as at the pipe. This indicates that the persistent and definitely timed vibrations of the reed can influence the volume of air within the pipe to vibrate at other than its normal rate. We may deduce from the foregoing facts the law that a volume of air, if exposed to individually slight and more or less heterogeneous vibrations, responds always with the production of its fundamental tone, but that persistent and well-marked vibrations of a definite frequency may impose upon it a rate of vibration which results in a tone somewhat different in pitch from its normal tone. Vibrations thus imposed upon a sounding body are called forced vibrations. Forced vibrations are of great importance in many musical instruments but are naturally of minor interest in the organ which consists of a series of what are practically resonators. The point to be emphasized is that the reed pipe illustrates the fact that vibrations may be forced upon a resonator.

If a tuning fork is struck and held free in the hand the tone emitted is feeble. But if the vibrating fork is rested by its lower extremity upon the surface of a wooden table the sound becomes much louder. This is true whatever the pitch of the fork. Here the table functions as a sounding board.

Forcing vibrations upon a body which has a periodicity different from that of its mass and shape is only practicable to a useful degree when the body thus caused to vibrate is sensitive to tones of widely varying pitch. There are numerous illustrations of this principle in musical instruments. It will be readily seen that the sounds of a musical instrument must be equally effective throughout the range of the instrument.

What a piano-maker or violin-maker must sedulously avoid is a response on the part of his instrument by vibrations of its own periodicity. For it would be fatal for the proper rendition of music if the piano or violin should unduly emphasize or fail to furnish any single tone.

A resonance-box or resonance-chamber is constructed to give sonority, that is, to vibrate with a large number of tones of widely varying pitch. In one of its commonest forms it consists of a thin layer of specially selected wood laid over or enclosing a chamber that contains air. The body of the violin and the sounding board of the piano are resonance-boxes. Less perfect resonance-boxes are the resonant air-chambers of the human mouth and nasopharynx which give a sonority to voice-sounds through a wide range. The conditions are, however, here much more complicated. The mouth might be regarded with equal propriety as a resonator which, by change of the size of the air-chamber and of the width of its aperture, can give reinforcement to many tones.

Any flat, broad, and thin body of elastic material that is free in the air to such an extent that its vibratility is not materially impaired by contact with solid bodies may be made to vibrate with forced vibrations. For example, a bed with woven wire mattress reinforces the sounds of the watch under the pillow or of the heartbeat. The sound of percussion is louder when the back of the subject is pressed against a door. And, of course, the perception of sound in general is mainly dependent upon the vibrations of the eardrum.

A resonator in the strict sense is an instrument designed by the physicist for the purpose of analyzing a sound into its individual tones or for determining the pitch of a tone. The resonator is usually of metal and of a globular, cylindrical or conical shape; for deep tones long tubes are sometimes used. A properly constructed resonator responds strongly only to a single tone of a definite pitch, so long as no alteration is made in the size of its volume of air or in the width of its orifice.

Any air-chamber with more or less elastic walls is a resonator in the sense that when a tone is sounded which is of the same periodicity as that of the vibrations of the volume of air contained within the air-chamber, it will respond to that tone with especial force. What may be called natural resonators,—that is, those in which the pitch has not been fixed artificially but is an accidental characteristic,—not being perfect acoustic apparatus may make a certain response to other tones, but that response is only for tones of nearly the same pitch as that of their fundamental tone and becomes greater the more closely that tone is approached. The thoracic cavity is such a resonator.

Harmonics are overtones that bear a simple arithmetical relation to the

fundamental tone. They have twice, thrice, four times as many vibrations per second and so on through a long series. It is the overtones that give a tone its characteristic timbre or quality. When the periodicities of a series of overtones are represented by even numbers the tone has a mellow quality, as that of the flue pipe of the organ. The reed pipe has a tone of a brilliant quality, its series having odd numbers. Besides the harmonics there are many other overtones called upper partials, some of which are more or less discordant with the fundamental tone. Such overtones are not produced with any uniformity; just what overtones will be formed, or will be prominent in a given sound depends upon many obscure and delicate conditions. They vary with the different conditions of individual resonators (the term being used in its wider sense) as respects the shape, the material of which the walls are composed and their hardness, smoothness and elasticity. These overtones give the characteristic qualities of sound by which, for example, the ear is able to distinguish the sound of the amphoric echo in a cask from that of a jug. The higher pitched the overtones, the fainter they usually are. It is important to note that, as the names given them imply, all overtones are based upon the fundamental tone of the cavity in which they originate and that they also may experience reinforcement, occasionally even becoming more prominent than the fundamental tone.

It is necessary to distinguish sharply between the action of the resonator and that of the resonance-chamber. The former, as has been remarked, responds normally only with its fundamental tone. If a sounding tuning fork is held near the mouth of a glass or metal cylinder with closed lower end, the length of which is one-fourth that of the length of the sound waves produced by the fork, the cylinder acts as a resonator and gives forth a loud musical tone of the same pitch as that of the fork. Why is this tone louder than that of the free tuning fork? There is, of course, no way of increasing the energy of the vibrations when once the fork has been made to vibrate. But the vibrating arms of the fork, which fly rapidly back and forth, do not impart the most suitable vibrations to the air which carries the sounds to our ears. But although only a small fraction of the sound waves from the fork enter the cylinder, the energy which creates the sound waves within it is applied to so much greater advantage, so far as the systematic agitation of the air is concerned, that the sound is considerably louder. It is customary to speak of the action of a resonator as a reinforcement of the original sound, as if there had been an actual increase in energy, but the fact simply is that the sound waves have encountered more favorable conditions for propagation to our ears.

In the resonance-chamber, or sounding board, on the other hand, we have broad and very vibratile surfaces which from their shape are adapted to impart their vibrations to the surrounding air to the best effect. Again there is no increase in energy but the existing energy is utilized to the best advantage for the production of sound. A violin string stretched in the free air emits when struck a feeble and poor sound, but the sound of the same string is loud when it is stretched over the body of the violin.

The word "resonance" is loosely applied to the action of both resonators and of resonance-chambers. When we speak of the pulmonary resonance of percussion we mean the sound of the fundamental tone of the thorax (combined of course with a number of other tones) from the free vibrations of the thorax as a resonator. But vocal resonance means the transmission of sounds, not for the most part of the pitch of the fundamental tone, which have been forced upon the thorax and, in the case of the violin, the "resonant" sounds result from vibrations that have been forced upon a resonance-chamber. It should be added that when a hall "resounds" to the sound of music or to the voice of a speaker the effect is produced by echoes; that is, the reflection of sound waves from the walls.

THE TRANSMISSION OF SOUND THROUGH THE CHEST

The study of the percussion sounds that are transmitted through the chest to the ear of a second observer is of little practical importance as a means of physical diagnosis but yields information of value as to the conduction of sound. One examiner percusses gently in an intercostal space anteriorly while a second listens at the back of the chest on the same side of the body. The gentlest blow imaginable, even one practically inaudible to him who percusses, is heard distinctly over all parts of the chest. But it is interesting to note that while the sound is not so loud near the base of the lung it is louder in the same intercostal space where it curves upward in the back than it is on the horizontal level of the part percussed. This fact corroborates the view that the major portion of the sound is conducted by the ribs.

But while percussion directly upon the ribs is louder than percussion of the adjacent soft parts the difference is less than one might expect and percussion in the middle of a wide intercostal space is carried almost as well as by the ribs. Hence we conclude that there is the freest interchange of sound between the ribs and the soft parts about them and that

wherever a pleximeter-finger is placed in the proximity of the ribs these bones will conduct a very considerable portion of the sound around the chest. The sound is not conducted chiefly in a straight line through the chest. For if it were thus conducted the heart would cast an acoustic shadow; that is, percussion over the heart would be nearly, or quite, inaudible over the back, so that it would be possible to outline the heart by auscultation posteriorly. This, however, cannot be done; the percussion-stroke over the heart is practically as distinctly heard posteriorly as that over the lung on ordinary light percussion which, of course, in view of the ease with which sound waves pass around small obstacles, is to be expected. On the other hand, there is some evidence that the presence of the heart does make some difference in the passage of the sound waves from the front to back in the chest, for Goldscheider states that he was able to outline the projection of the heart upon the posterior surface of the chest by auscultation of very gentle percussion over the heart, the note of which changed when the percussion passed beyond the heart borders.[1]

In such experiments the lightest possible percussion is essential. A stronger blow would call forth the resonance of the chest and obscure the results. In other words, the progressive sound waves proceeding from the pleximeter must not be allowed to become the stationary waves of the thoracic resonance.

Goldscheider made experiments upon the cadaver which show that the sound of percussion is conducted around the chest in its wall but also to a lesser extent passes through the lung. He also percussed a lung removed from the body and found that when the lung was inflated the lightest percussion could be heard from the top to the bottom of the lung.[2]

In the case of the lung percussed through the chest wall the conduction is, of course, not solely that of sound waves. The force of the blow itself disturbs the equilibrium of the structures that lie in its path so that the sound produced is due to some extent to the concussion of the parts. This is shown to be true by the fact that a noiseless pleximeter of soft rubber gives satisfactory results in the percussion of the chest, which shows that the vibrations which are capable of exciting the action of resonators need not necessarily be sound perceptible to our ears.

The question has been raised whether it is possible to give direction to the vibrations that proceed from the pleximeter—especially, of course, whether the vibrations penetrate the lung more deeply in the direction

[1] Goldscheider, *Deutsches Arch. f. klin. Med., Leipz.*, 1908, XCIV, 480.
[2] *Loc. cit.*

of the axis of the blow than elsewhere—so that the percussion can be localized. Opposed to this conception is the fact that, in general, sound proceeds in spherical waves. But according to Martini extension of sound in spherical waves is preserved exactly only in isotropic media and then only strictly when the source of sound is spherical,—that is, when its vibrations are of radial symmetry. These conditions do not, of course, obtain in percussion of the lung. Martini made experiments that do not confirm the existence of a direction of impulse in the lung. Really, however, the important question is not whether sound waves can be sent in a given direction through healthy lung but whether they can be returned from lesions—that is, from regions of marked heterotropism—through healthy lung in such a way that the presence of said lesions becomes clinically recognizable. There is no question that in spite of the extraordinary diffusion of sound by the chest wall this is practicable to the extent that alterations of note betray the fact that the vibrating lung has less depth over such a lesion than elsewhere. If this were not the case localizations of deep lesions would be impossible. This subject is referred to under Percussion.

Since, as has been stated, the ribs pass upward in their course backward so that the vibrations of percussion or other shock communicated to the chest wall are carried preponderatingly upward around the chest, and since the upper lobes underlie directly the anterior surface of the chest, it follows that the vibrations from a commotion of the anterior thorax, whether carried around or penetrating the lung, implicate chiefly the upper lobes.

On the other hand, the vibrations of a percussion-blow upon the back over the lung below the level of the third dorsal spine are carried downward as well as forward by the ribs, and if they reach the upper lobes at all reach them anteriorly near the base of the lung, in a region where damping by the heart, liver and stomach are of influence, while that part of the vibrations which penetrates the lung reaches directly only the lower lobe. Hence it may be said that the structure of the thoracic walls and the arrangement of the lung lobes both tend in a general way to limit vibrations which are communicated anteriorly to the upper lobe and posteriorly to the lower lobe. The middle lobe of the right lung occupies a neutral position in this regard. These considerations are of some importance in view of the fact that the visceral pleura which covers the individual lobes probably has considerable effect in limiting and reflecting the vibrations of the masses that it contains. The structure of the thorax and of its contents, in short, does not favor but rather tends to oppose the general dissemination throughout the lung-mass of vibra-

tions communicated to any part of it. The effect of the arrangement of parts is such that, without the provision of rigid barriers which might in themselves on occasion prove a source of danger, one portion of the lung is to some extent shielded against the extension to it of vibrations of dangerous intensity which might involve another part.

Anteriorly the ribs terminate in cartilage which, excepting in the case of the first rib, articulate with the sternum by means of joints. The sternum, an elastic bony plate, is composed of several pieces, some of which are late in uniting with one another by bone; others never entirely lose their cartilaginous structure. The mode of combination is such as to tend to minimize the proper vibrations of the sternum as a flat and relatively thin bony plate. In general, the anatomical structure of the anterior part of the thoracic wall is calculated to diminish the conduction of vibrations from one side of the body to the other across the median line. This is of primary importance in minimizing the shock of concussion, but necessarily affects also the transmission of sound waves.

Posteriorly, again, the ribs articulate with the spine, which articulation tends to diminish the transmission of vibrations. Nevertheless some vibrations reach the spine through both ribs and soft parts. Now, the spine is heterotropic acoustically; that is, it is composed of heterogeneous sound media, the bodies of the vertebrae and the interposed cartilages. Vibrations will not, therefore, be readily transmitted up and down the spinal column. The individual bodies of the vertebrae, from their structure, are capable of communicating sound fairly well laterally, but here again, the small size of the individual bones and their structure will favor the transmission of vibrations of relatively high frequency only. The effect of too violent commotions of a single rib upon the individual vertebrae is halved in the case of most of the ribs by articulation with two vertebrae. Hence we find in practice little evidence of transmission of sound that arises in one side of the thorax to the other side by means of the spinal column, save under exceptional conditions during disease, when the vibrations thus transferred are of a much higher frequency than that of the normal lung vibrations. Anteriorly to the spine the mediastinal structures damp powerfully vibrations that seek to pass them.

It appears, therefore, that not only is there provision for preventing too ready communication of vibrations from one lung-mass to another of the same lung, but also that the vibrations of one side of the thorax are damped in various ways so as not to pass freely to the other side. And, as we saw in the first instance, so in the second the barriers are not absolute; there is a gentle checking of vibrations at many points, nowhere are they definitely turned back upon themselves. All of these

provisions are undoubtedly of great importance in enabling the thorax to withstand with minimal injury to its contents and to its structure the shock of falls or other extreme commotion. Teleologically, the arrangement of the thoracic structures is useful chiefly for severe aperiodic shocks, but it is also of considerable importance with reference to the less dangerous vibrations which we call sound. It is evidently not desirable that strong communicated vibrations shall ever cause the entire thorax as a single whole to vibrate powerfully to its fundamental tone.

The lungs on the whole are fairly well isolated from each other, coming nowhere into immediate contact and only nearly approaching one another at their anterior edges.

We may therefore safely conclude that the thoracic cage does not vibrate in its entirety, in a manner analogous to the walls of a resonator, in response to a commotion which involves one only of its halves and, therefore, that the effect of percussion is practically confined, as a rule, to one hemithorax. However, a certain influence appears to be exerted upon one half of the thorax from the other half when there are morbid changes of volume in one or both lungs, a point which will be discussed elsewhere. Experience shows that the thorax, as a whole, does not function as a resonator, for if it did it should yield its fundamental note over all portions of its surface; we should be able to hear the sound of percussion as well over the lung of the other side as at the point percussed which is far from being the case, the limitations of percussion sound being very apparent even as respects the different portions of the side percussed. It is customary to assume that the pitch of the fundamental lung-note is determined by the volume of air contained in the hemithorax. A comparison of normal lung-resonance, with tympanitic resonance, however, shows that this cannot be correct without at least some reservations. In tympanitic resonance of the stomach and intestine the percussion-note obtained consists preponderatingly of the fundamental tone of the volume of air within the cavity percussed. Without question the air here vibrates as a whole. If the stomach is partly filled with liquid, this liquid will affect the tone in that its presence diminishes the volume of air, but percussion does not reveal the height of the liquid; on the contrary, percussion gives the same tone over the liquid as over the gaseous contents of the stomach because the tone produced is due to the vibrations of the volume of air and of the walls of the cavity that contains the air. If the lung vibrated as a whole under the percussion-stroke, we should obtain the same note over a localized focus of disease as we do over adjacent healthy tissue, in which event topical diagnosis would be impossible. This is shown to be true from the

results of percussion over lung which has a definite tympanitic resonance; in such a lung, or portion of a lung, dullness cannot be localized because the part concerned vibrates as a whole. It has been found by Goldscheider and by other experimenters that pieces of the lung yield on percussion tympanitic resonance the pitch of which is lower in a larger than in a smaller piece and is therefore chiefly dependent upon the volume.

Light is thrown upon the vibrations of a lung, or a piece of a lung, which is in a state of relaxation, by the study of foaming liquids as transmitters of sound. The following is abstracted from the work of Geigel.[3] If one taps upon the bottom of a glass cylinder full of a foamy liquid (beer or carbonated water) a deep tone is heard, much deeper than the tone of the empty cylinder. As the bubbles ascend and break and the froth disappears from the surface, the sound of percussion grows progressively higher in pitch; when bubbles are no longer given off the glass filled with the dead liquid yields no sound or only the sound of the glass. It can therefore only be the intimate mixture of small portions of gas and liquid which, vibrating, produces the deep tones. Experience shows that sound is not reflected by small surfaces. In such small dimensions as those of the individual bubbles and the liquid that surrounds them there can, therefore, be no reflection of the sound waves at the bounding surfaces; the sound waves slip through without any reflection. It must be that such foamy liquids vibrate as a whole, that reflection takes place only at the borders of the whole foamy mass, for there the dimensions of the border proportionally to the length of the sound waves are great enough. The tone is deeper than that of an equally large air-space of the same shape because the mass is greater, for foam, since it consists not only of air but also of much heavier liquid, is without question heavier than air. We have then a relation similar to that of two equally long and equally stretched strings, one of which is heavier than the other. The heavier when struck yields the deeper tone. The greater the volume of the vibrating body—the foam—the deeper its tone. It is clear that a body vibrating so sluggishly will damp the much more rapidly vibrating parts of the glass walls and will not let the high clang of the glass appear. This experiment is fundamental for the explanation of the percussion-sound of the lung that is not under tension. The lung, like the foam, consists of small air-spaces surrounded by thin walls of a heavier substance, the dimensions of the air spaces, or cells, being much too small to reflect sound. The greater the volume of the lung and the greater consequently the size of the cavity that contains it the deeper the tone.

[3] R. Geigel, "Leitfaden der diagnostischen Akustik," Stuttgart, 1908.

Martini's experiments show that when a lung inflated to a pressure corresponding to its physiological tension during life is divided, as a vibrating body, into two portions by the pressure of a clamp, each portion has a percussion-note of the same pitch as that of the entire unclamped lung, provided that neither of the parts is very small—each part should have a depth exceeding 4 cm. These results he interprets to mean that the lung does not vibrate as a whole when percussed. He says: "In media of such heterotropic and porous structure (as the lungs) the relative movements of the individual vibrating portions are of a such magnitude that they may not be disregarded in comparison with the movement of the continuous mass. In the whole system independent vibrations of individual systems are possible. As soon as the advancing wave (of percussion) has imparted to the individual partial masses the maximum of their acceleration by virtue of their elastic force, which is here proportional to their tension, they will strive to regain their position of equilibrium about which they thus vibrate periodically. The sound thus arising will be purely dependent with respect to its rate of vibration and its damping upon the elastic force, the mass, and a friction factor of the vibrating parts. On the other hand, the frequency of vibration will be in no way dependent upon the strength of the percussion-blow. This factor has only influence upon the loudness, first, by increase of amplitude of the vibrations; second, through the commotion of a greater number of vibrating portions." Martini [4] corroborates these views by experiments which show that two lungs, properly inflated, and placed in closely fitting contact in a box give a percussion-note of the same pitch as that of one of the lungs tested separately in the same way.

The facts regarding tympanitic resonance show that it is not the heterotropic structure of the lung, nor the sluggishness of vibration of the lung-tissue as such that produces such a result, but the lung tension. The normal lung, when physiologically tense, obstructs and retards to a certain extent the passage of the vibrations peculiar to the space that it occupies so that, while the sound passes as progressive waves through the entire mass with ease, there is sufficient intrapulmonary reflection of sound to create stationary sound waves which represent the vibration of a part only of the lung.

But though there seems to be no escape from the view that percussion of the lung is always or usually localized in its effect, the fact remains that the lung-note proper is always of approximately the same pitch for the individual healthy lung and that that pitch is the same as that of vesicular breathing. Likewise, the same pitch represents the optimal

[4] Paul Martini, *Deutsches Arch. f. klin. Med., Leipz.*, 1922, CXXXIX, 65.

pitch for producing fremitus in that same individual if he has a deep voice. We may say then that the only sounds which pass with readiness through the air-cells of the healthy lung are sounds of, or near to, the frequency of its proper vibrations.

We have here the special case of a body which occupies a resonator and yet has its own specific rate of vibration, a rate nearly or quite the same as that of the resonator. If the vibrations are produced locally, as by the blow of percussion, and are reflected sufficiently to produce an audible tone before traversing the entire lung, these local disturbances under the conditions will nevertheless have a fundamental tone of the same pitch as that of the hemithorax vibrating as a whole—so far as the production of the lung-note is concerned, the part is equal to the whole. That the structure of the lung explains the relative sluggishness of its vibrations and their tendency to local reflection may be readily conceded; it is not so apparent at first sight why the specific vibrations of lung-tissue should be of the same pitch as that of the cavity of the hemithorax.

The resonator of the physicist has walls of unchanging tension and the contained air is never under positive pressure. But when the air-space is a closed space and its walls experience changes of tension, such tension influences strongly the production of the normal rate of vibrations. This is very simply illustrated by the distended buccal cavity. If the slightly distended cheek is snapped by the finger nail a tympanitic sound is produced. Continue to snap, and increase the pressure very gradually. The tympanitic note becomes fainter and fainter and seems to be higher in pitch; finally nothing is heard but the impact of the finger against the tense cheek. Vibrations of some kind are still produced, no doubt, in the contained air, but they are relatively restrained and do not pass through the walls of the cavity to produce sound audible to our ears. The law of the vibration of the air of a cavity with the production of its fundamental tone must, therefore, be modified to except cavities in which the air is under tension and of which the walls are too tense to vibrate. These observations apply to the lung which if strongly distended fails to yield its characteristic note on percussion. The pressure requisite for this, however, never obtains during life. But when the lung is expanded to its physiological degree the external membranes and the internal partitions, which, relaxed, had been no obstacle to vibration, now prevent the production of tympanitic resonance and reflect in part locally the sound that before had passed so freely through them. The end attained by this reflection of vibrations is the same as that sought in the anatomical relations of the thoracic structures—a limitation of generalized vibrations.

A severe commotion, not instantly destructive, which is imparted to the thorax, if it is passed through the lung as through a homogeneous sound medium would cause the latter, inevitably, to vibrate violently at the rate peculiar to the air-containing cavity. By partial reflection these vibrations are turned back—not arrested—here and there through the lung. Its whole mass may thus be saved from dangerous vibration as a single vibrating body. This is an additional illustration of the fact that in many ways and at various places commotions of the thorax are dispersed and guided as to direction, but are nowhere opposed by inflexible barriers.

The apparently mysterious fact that the specific rate of vibration of localized lung-masses under normal tension is the same, or nearly the same, as that of the fundamental tone of the thorax when it is filled with the physiologically expanded lung, is to be explained by the governing influence of the contents of the cavity upon the vibrations of its walls. It is a law of resonator action that, while the contents and the walls of a resonator must both be able to vibrate and must vibrate in unison, the vibration of the contents, being determined, as a rule, by the volume of contained air, control the vibrations of the walls. The specific rate of vibration of portions of the lungs must be due to the adaptation of their structure to vibrate with the deep vibrations characteristic of the entire lung-mass which could be affected by a thickening or stiffening of the solid portions of the lung. If there were not some such provision, a subdivision of the lung having a smaller volume of air would necessarily have a more rapid rate of vibration than the whole lung.

At the same time the rate of vibration of lung-tissue has the closest connection with the normal rate of vibration of the thoracic cavity. In Martini's examinations of isolated lungs he obtained a rate, in the individual case, that differed with the size of the lung—a low rate in men, a higher rate in the smaller lungs of women. And since he showed that the râle of large subdivisions of the lung is the same as that of the undivided lung, it may be inferred that the specific rate of lung vibration likewise varies with the individual lung and in the same person with the increase of the lung in size.

The lung of the young child is, no doubt, as well adapted physiologically and anatomically for the performance of its functions as that of the adult. In fact the high elasticity of its tissues and of the thoracic walls fits it more perfectly for resonator action than that of the adult. We do not know whether its specific rate of vibration is an acquired or innate characteristic, but there can be no question that, in the process of growth, that vibration rate will require to be changed constantly to agree with the changing normal rate of vibration of its containing

cavity. If there should be a discordance it would probably consist in the persistence of a rate of vibration of the lung more rapid than that of the growing chest cavity. In this case the delicate tissues of the lung would be subjected to strains and these strains would naturally be met on the part of the organism by a thickening and strengthening of the tissues. This cannot take place in the air-cells for nothing must be permitted to interfere with their function.

The only part of the lung that may be strengthened without injury is its framework, the fibrous sheaths of lobes and lobules, and the fibrous coats of tubes and vessels. Such thickening will lead to a slower rate of vibration on the principle that a heavy or weighted string vibrates more slowly than a light or unencumbered string of the same length. With the well-known economy of nature such compensatory changes will, physiologically, not be in excess but will be limited in extent to the actual necessities of the case; that is, the lung will become tuned to the note of its cavity. And, while thus becoming tuned, let us suppose farther that, on account of the great flexibility of the child's thorax and the elasticity of its lung-tissues, vibrations imparted to the lung traverse it with undue violence and cause a commotion of the entire lung to an unwelcome extent. The remedy will be a thickening of the fibrous sheets most exposed to such strains, which will prevent undue stretching and make the partitions more competent to check and, checking, to turn back partially—not to arrest—the vibrations. Such thickening, once more, will not be in excess, but just sufficient for the purpose. That is to say, the parts thus protected will be brought to vibrate in harmony with the dominant vibrations of the thorax. The part will thus come to have the same rate of vibration as the whole. Such adaptation is to be looked upon as the adjustment of the lung, as a vibrating complex, to the strain to which it is habitually exposed, namely, the proper vibrations of the cavity in which it is situated.

To repeat, a too severe rate of vibration of the lung is guarded against by a thickening of fibrous planes and bounding membranes which increases as the growing thorax develops a deeper tone and, consequently, exposes the lung-tissue to a commotion from vibrations of greater energy; so that at all times the lung-tissue in health is, as it were, attuned to the vibrations to which it is most subjected and, at the same time, is also prepared to resist an undue violence of these normal vibrations.

In the foregoing pages have been discussed the modes of reaction of the thorax and its contents to commotions imparted to it locally. It remains to consider more specifically the relation of the thoracic wall to the blow of percussion. We are concerned now with the analysis of

the sound that reaches our ears. When a percussion-blow is struck upon a pleximeter placed upon the normal chest wall we hear a sound which contains both low and high-pitched tones. The high-pitched tones are produced by the localized concussion of the chest wall. They are compounded of the sounds of the struck pleximeter, namely, the sound of the impact of the hammer, of the vibrations of the pleximeter and also of the vibrations of the chest wall. These latter vibrations are due almost entirely to the ribs and will be the louder the more nearly the pleximeter approaches the rib and loudest when it rests against a rib. With finger-finger percussion the sounds of the pleximeter seem to be weak. But if the person percussed has a trained ear he perceives distinctly the change of note effected by increased proximity to the rib. Over some parts of the chest, where the intercostal spaces are narrow and the ribs if not actually touched are near to the pleximeter-finger, the sounds seem hard in quality and high pitched. Where the pectoral muscles cover the bones or where the intercostal spaces are wide the sound seems soft and low pitched. The soft parts of the chest while, as has been remarked, they transmit sounds well, do not give off audible sound. Now, the sounds of the pleximeter and of the chest wall that we hear are given off directly to the surrounding air from the structures implicated by the blow. The vibrations of the chest wall are also communicated to the underlying lung. These vibrations pass directly through the lung and are dispersed in all directions, reaching all parts of the lung and the structures adjacent to it—a fact which is determined by auscultation upon the opposite pole of the percussed diameter of the chest and over adjacent vertebrae, etc. Writers make a distinction between the sphere of the blow and the sphere of acoustic effect. Let us suppose that the sound waves which are forced upon the lung from the pleximeter undergo reflection, become stationary sound waves and produce sound in the first of the subdivisions reached which is capable of reflecting sound. These stationary sound waves, which are excited by low-pitched elements of pleximeter vibration in accordance with the specific vibratility of the part of the lung concerned, have a frequency the same as that of the vibrations of the resonator within which they have been formed. The phenomenon therefore represents resonator action limited, so far at least as the production of audible sound is concerned, to a portion of the resonator. The resonance manifests itself by a deep-pitched tone which passes through the chest wall causing it to vibrate and, in so doing, to create vibrations of the same frequency in the air which surrounds the thorax. The sound heard by the examiner is, therefore, a combination of the high-pitched sounds given off

by the pleximeter and the chest wall and of the low-pitched tone of lung-tissue. In the case of the high-pitched sound the sound waves have traveled directly to the ear while the low-pitched sound is the result of the sound waves traversing the sound-producing sphere at least twice, but on account of the velocity of sound the two series of sound waves seem to reach the observer's ear simultaneously. The sphere of acoustic effect is that portion of the lung within which stationary sound waves have been formed that have sufficient energy to pass through the chest wall and arrive as sound at the ear of the observer who percusses. The blow wave, however, has not been limited to the sphere of acoustic effect. The sound waves (which it must be remembered are merely one class of the vibrations excited by the commotion of the chest) pass on to other acoustic subdivisions of the lung. It may be that stationary waves are also excited in these deeper subdivisions, but too feebly to return to the listening ear. In any case the sound waves, spreading in all directions, advance as progressive waves to the opposite side of the chest there to be reflected and dispersed and to die out. Under the definition given the fact that another observer might pick them up on that surface as sound would not justify the extension of the sphere of acoustic effect to include such waves; they belong to the sphere of the blow.

Since in percussion the hemithorax does not vibrate effectively as a resonator throughout its entire extent, one speaks of forcing the vibrations of the pleximeter upon the lung, although the result is practically the production of the fundamental tone, so that the process really amounts to resonation. Similarly, the passage of the stationary vibrations of the lung outward through the chest wall is regarded as that of returning sound waves (so that the action is called retrograde action) which force themselves upon the wall, although, as the wall of a resonator vibrating to its fundamental tone, its vibrations are really free vibrations. The thinner and more elastic a structure the more readily are vibrations forced upon it. The more emaciated the chest, therefore, the stronger the lung-note. It is well known that the chests of powerful and well-muscled athletes are relatively dull on percussion, which is of course due to the fact that their firm and thick walls are not readily caused to vibrate to sound waves that pass through them in either direction. Willed tension of chest muscles may have the same effect. Some English authorities at one time held the view that the lung-note of percussion was due to the vibration of the chest wall, for the reason that strong contraction of the thoracic muscles produces dullness. Morbid conditions, spasms of chest muscles and deformities of the bones of the thorax, also play a part in

impairing the transmission of sound through the chest wall and in diminishing its own vibrations.

Changes of Pitch of the Percussion-Note as a Diagnostic Sign, Especially of Cavity

When solidification of the lung extends to a large bronchus, or a solidified apex conducts sound well to the trachea, percussion may yield a tympanitic note due to the resonator action of the tube that is reached. In tympany thus produced, if the patient's mouth is open during percussion, the note will be lowered in pitch when the mouth is closed and will be lower still if both mouth and nostrils are tightly closed. This marked change of the percussion-note when the air-passages are cut off from communication with the outside air is known as Williams' tracheal tone. The phenomenon sometimes enables a distinction to be made between tympany due to consolidation and the tympany of a relaxed lung, the changes in pitch when the mouth is opened or closed being greater in consolidation than in relaxation.

Wintrich's sign of cavity is a change of the percussion-note over the region of the suspected cavity to a lower pitch when the mouth is closed. In this case the change in pitch is not so marked as in the "tracheal tone" for the air-tube is now in communication with a large cavity, is therefore practically equivalent to a pipe connected with a resonator and the larger volume of air in the latter governs the pitch. According to Gerhardt,[5] experiment shows that the percussion-note of a cylindrical column of air gives a much greater difference of pitch when one end of the cylinder is closed than that furnished by a cylinder of the same dimensions to which a much larger sphere has been soldered. A marked lowering of pitch when the mouth is closed speaks, therefore, against the diagnosis of cavity [6] and in favor of Williams' sign. A point of distinction is, as Kuthy points out, that Wintrich's sign may be brought out over a superficial cavity by very light percussion, while relatively heavy percussion is needed to elicit the tracheal tone. Percussion of the trachea in health yields a lower tone when the mouth is closed.

It is easy to percuss the side of one's own larynx. With tightly closed glottis the note is dull. It becomes louder, more resonant and higher pitched when the glottis

[5] Gerhardt, "Auskultation u. Perkussion," Tübingen, 1900.

[6] Kuthy and Wolff-Eisner, "Prognosentellung bei der Lungentuberkulose," Berlin und Wien, 1914.

is open and these characteristics are still more marked when the mouth and glottis are both widely open. As in Gerhardt's experiment, the cylinder (larynx and trachea) when it communicates with the resonator represented by the cavities of the mouth and nasopharynx, is governed as to its tones by the resonator. The combined cylinder and resonator have a higher percussion-note when the orifice of the resonator is open than when it is partially closed by closure of the mouth, the nostrils remaining open. And when the nostrils are also tightly closed the note becomes still lower pitched. These findings are in agreement with the behavior of a resonator the size of the orifice of which can be changed at will. Contracting such an orifice leads to a lowering of the pitch of the resonator. In seeking for Wintrich's sign or Williams' tracheal tone a more marked difference in the pitch of the tones could be obtained if the patient, instead of simply closing the mouth, shuts off the resonator from the cylinder by closing the glottis—that is, by holding his breath. In cavity cases manifestly the cylinder is connected with *two* resonators.

An "interrupted Wintrich"—that is, the presence of the sign when the patient stands and its disappearance when he lies, or vice versa—is evidence of a cavity containing considerable liquid, in which the opening into the bronchus is obstructed in certain positions of the body.

Friedrich's sign of cavity is a tympanitic note which becomes higher in full inspiration. But if the size of the cavity could be increased in inspiration, its note should be lower. Elevation in pitch must be due to increase of tension which tends either to diminish pulmonary resonance, or, if it acts upon the cavity, to change tympanitic into atympanitic sound.

The strength of percussion is all-important in such studies. Practically, to elicit the true fundamental note of the thorax when respiratory muscles are tense, strong percussion is necessary. Failure to do this has led to misapprehension. Thus, Traube, according to Selling, found a rise in pitch in the low tones in percussion during inspiration except in the case of a convalescent typhoid patient, which Selling explains by the assumption that he used much too light percussion and only found the true condition in a thin individual in whom light percussion was sufficient.[7] Similarly, Von den Velden, auscultating over the back the sound of light percussion upon the front of the chest, finds that the pitch is raised when percussion is performed while the breath is held after full inspiration, although the thorax being increased in size the fundamental note, if changed, should be lower in pitch.[8] The rise in pitch noted with the strength of percussion used is due to a relative increase of the bronchial component which becomes more apparent when the vibrations of the chest wall are checked by the increased tension of the

[7] Selling, *Deutsches Arch. f. klin. Med., Leipz.,* 1907, XC, 163.

[8] Von den Velden, "Die Klangleitung der Lunge," *Deutsche med. Wochenschr., Berl. & Leipz.,* 1905, 581.

respiratory muscles and a greater obstacle is opposed to the passage outward of the low-pitched tones.

If an oval cavity partly filled with liquid has its long diameter parallel with that of the body, the percussion-note over it will be higher in pitch in the upright position than when the patient's body is horizontal; but if the long diameter of the cavity is at right angles to the axis of the body, these changes of position will have the opposite result because the column of air within the cavity will be at its longest when the patient is erect. This is known as Gerhardt's sign.

The above explanation of Gerhardt's sign is the current one, but to one who has read the preceding pages it should be evident that it must, as a rule, be incorrect: for, the length of the longest diameter governs the pitch of the sound only when the disproportion between this dimension and the other dimensions is considerable, as in tubes. Cavities are usually approximately spherical or oval in shape; in such cavities the volume of air determines the pitch, and that volume would not be affected by changes of position. Geigel points out that usually the sound becomes higher when the patient sits, which he ascribes to increase in tension from the traction downward of the liver in that position, the same change being sometimes apparent in the tympanitic note of an infiltrated portion of the lung when there is no cavity. The reverse change —the sound is higher when the patient lies—is very rare. It is possible, as Geigel says, that the percussion-note of such a cavity might be altered by changes in its tension due to the displacement of liquid; this could never occur from changes in the shape of the air-space.

Gerhardt's sign is evidently of more interest as a problem in the acoustics of the chest than as a guide in diagnosis, and the same remark applies with almost equal truth to the other signs based on changes in the percussion-note.

That the breath-sounds originate in the larynx is shown by the fact that when the rima glottidis is opened wide by the willed contractions of the laryngeal muscles all sounds, both of inspiration and of expiration, disappear. Place a binaural stethoscope against the side of your larynx and study the sounds of normal respiration. The sounds are harsh and—many of them—high pitched. Then, opening the mouth wide and trying to hold the throat open, take a deep inspiration as noiselessly as possible. It will be found that during such an inspiration the laryngeal sounds diminish very considerably and you may be able to distinguish the vesicular murmur. With some practice it should be possible to eliminate all sound during at least an occasional inspiration. When the larynx is silent the lung is also silent, but if there is the slight-

est sound in the larynx another observer, listening over the lung, will detect a sound at his place of auscultation. The experiment, if successful, will demonstrate the complete dependence of thoracic upon laryngeal sound.[9]

The glottis is normally expanded during inspiration by the contraction of the posterior crico-arytenoid muscles which rotate the cricoid cartilages outward and thus separate the vocal cords, the movement being synchronized with the other respiratory movements as an integral part of the process of inspiration. When inspiration ceases the muscles relax and the cords return passively to their position of rest, which is the position of expiration. During both inspiration and expiration the vocal cords normally overhang the cavity of the larynx, so that they produce sound when a current of air passes between them in either direction. The sounds of respiration are caused by the vibrations of the edges of the cords, not by the vibrations of the cords in their entirety, as occurs when they are made tense in phonation. The edges of the more or less relaxed cords may be said to flutter in the air-currents. In so doing they do not produce a definite tone but a noise composed of a great number of tones, all of them weak. Sound waves are thus produced in great variety in breathing, but the breath-sounds, as we hear them, even over the larynx or before the open mouth, are not the sounds caused directly by the fluttering of the cords but are those original sounds greatly modified by the stationary sound waves which they have excited in resonators. The properties of resonators have been in part considered. An additional characteristic, which has not been dwelt upon, is that the pitch of a resonator is not determined solely by the volume of air that it contains, but also by the size of its orifice, the tone emitted being the lower the smaller the orifice. In the resonators that we have discussed the size of the orifice is not subject to change, but in sounds which have the mouth as a resonator the pitch of the tones is lowered by protruding and nearly closing the lips, thus increasing the size of the resonator and narrowing its orifice, and is raised by opening the lips to widen the orifice and retracting them to diminish the size of the resonator.

That the air-tubes also act as resonators is shown by the fact that the bronchial breathing of morbid conditions is not altered in pitch by changes in the force of respiration. Furthermore, the pitch of bronchial breathing lies in the range of pitch of sounds to which tubes of the size of bronchi of the second and third order respond as resonators, as

[9] G. E. Bushnell, "Mode of Production of the So-Called Vesicular Murmur of Respiration," *J. Am. M. Ass., Chicago,* 1921, LXXVII, 2194, and "The Discoverer of the Mode of Production of Breath-Sounds," *J. Am. M. Ass., Chicago,* 1923, LXXX, 895.

Müller has shown.[10] It is characteristic of stationary vibrations that, their rate being that peculiar to the volume of air concerned, their pitch is not influenced by changes in the amount of energy to which the vibrations are due. Variations in the force of the breath-sounds without other change in the conditions do not, therefore, affect the pitch of the breath-sounds. This statement is true of weak sounds, notwithstanding the fact that it is possible to change the tone of a pipe by increasing the force of the air-current. Great increase of force is necessary to produce even the first overtone, the octave. Such an alteration in pitch can never occur in the air-passages in connection with normal respiration. It is easy to convince one's self of the persistence of a fixed rate of vibration in laryngeal sound by a simple experiment. With half-open mouth make an audible sound, as of aspirate breathing, with the larynx. Practice this with the same force of breathing and the same position of the mouth until you feel sure that you can continue to produce the same sound, so far as the larynx is concerned, whatever the position of the lips. Then, continuing to breathe with the same force, purse the lips. The sound will become very noticeably deeper in pitch. Returning to the former position of the lips, the sound becomes, of course, of the same pitch as at first; but, opening the mouth more widely and retracting the lips strongly, the pitch rises still higher. Increasing or diminishing the force of expiration and of inspiration (for the experiments should be repeated in inspiration) makes no difference in pitch if the position of the vocal cords remains the same. In this experiment the noise of respiration, already altered by the resonator action of the thorax and of the air-tubes, comes under the influence of the mouth as a resonator. The tone of the noise that has the same pitch as that of the mouth undergoes reinforcement and becomes the predominating tone. When the size of the resonator and of its orifice is changed other tones are selected for reinforcement, but each tone remains constant as to pitch, irrespective of the amount of energy expended in producing the sound, so long as the position of the lips and of the vocal cords is unchanged. The experiment also demonstrates very well the fact that a noise is made up of a number of individual tones.

Sounds Produced by Movement of Air Through Tubes

It has been found by studying the sounds produced in tubes that, while sounds may be caused by vibrations which arise in the continuity of the

[10] F. von Müller, "Zur Physikalischen Diagnostik," *Verhandl. d. deutsch. Kong. f. innere Med., Wiesb.*, 1911, XXVIII, 184.

tube, they are produced much more readily and with much less force when the fluid (air, blood, etc.) passes from a narrow to a wider space. This applies to the constriction of the air-current effected by the vocal cords—we have seen how easily sound is produced in the larynx by even a slight movement of air—and it also applies of course to the emergence of air from a narrow to a wider air-tube. Only the slight force of normal respiration accounts for the fact that sound is not produced by the passage of air outward from the smaller bronchi into the main bronchi and the trachea. Similarly, we should expect that sound revealed by auscultation over the lung would be produced by the air that passes through an intubator tube in the larynx or through a tracheotomy tube, in both of which cases the air-current from the trachea is very considerably narrowed; and observation shows that such is the case. As we have seen in the case of the organ pipe, the pitch of sounds produced in tubes, or amplified by them as resonators, is determined chiefly by the length of the column of air. In view of the relative shortness of the branches of the bronchi the tones produced, or more usually reinforced, by the individual tubes would be expected to be high pitched. That this is true is shown by the sounds of bronchial breathing. Like other resonators, neither the wall of the tube nor its contents can vibrate alone; every motion which the one makes is at once imparted to the other and the formation of sound waves is only possible when each vibrates in harmony with the other. And this must be true even of the noises (or complex of tones) which are produced in the respiratory passages. We must conclude therefore that the bronchi, if they produce sound, will yield sounds or noises of relatively high pitch which cannot be changed by increased or diminished force of the air-current.

"Reciprocating breathing" is an illustration of sound produced in the bronchi, although the sound heard over the lung is not probably the sound as it is actually formed in the tubes.[11] If the glottis is closed and the thorax is elevated as in inspiration, the diaphragm being kept relaxed, the air in the lower parts of the lung and with it the diaphragm is forced upward by the atmospheric pressure. When the thorax is allowed to fall and the diaphragm is activated, the glottis remaining closed, the process is reversed; the lower parts of the lungs expand. Air can thus be drawn to and fro within the lung with considerable force. In this form of respiration which, following Hoover, we will call reciprocating breathing, respiratory sound is produced. The sound has not the "breezy" quality of the vesicular murmur, but has more of a bronchial character, yet its pitch is the same as that of the vesicular murmur. The quality

[11] Bushnell, *loc. cit.*

and loudness of the sound is the same in both inspiration and expiration as heard at any given place; its pitch is not altered by increase or diminution in the force of the respiratory movements. As for the mode of production of this sound; of course the air is alternately driven out of and sucked back into the bronchi of the various lobes and, since from the nature of the case, the outflow from one part of the lung must become the inflow of another, the amount of air interchanged not being increased or diminished, so long as the glottis remains closed and the force of the individual respiratory acts remains equal, expiration must exactly balance inspiration. Considering the lungs as a whole, both expiration and inspiration are proceeding simultaneously somewhere during both phases of the reciprocal interchange of air between upper and lower lobes, although at any given point—as at the point of auscultation—phases succeed one another which practically amount to alternate inspiration and expiration. During this process there is a constant passage of air from smaller to larger tubes and at the same time from larger to smaller tubes, thus affording every opportunity for the production of the vibrations of the bronchi which are no doubt the cause of the sound. Its unvarying and low pitch shows the influence of the thorax acting as a resonator. Here we have a small volume of air driven with considerable velocity through normal air-tubes. The experiment proves that, given a sufficient force and velocity of the air-current, sound can be produced below the larynx which will excite the resonation of the thorax. The sound is, of course, a noise like other respiratory sounds. While the fundamental tone is that of the thorax, what we have called the bronchial quality is probably due to higher tones. The experiment is interesting as showing the predominance of the thorax as a resonator over sounds produced in the bronchi which must vibrate with their own periodicity. Even under such conditions really high-pitched sounds do not pass through the air-cells.

The strongest argument for the intrathoracic origin of the vesicular murmur has been the apparent persistence of vesicular breathing over the lung when a tube has been inserted into the larynx, thus excluding the action of the vocal cords, or when the trachea has been opened, so that air no longer passes through the larynx. These phenomena are however simply additional illustrations of the fact that the passage of an air-current from a narrower to a wider space may create sound waves capable of undergoing reinforcement in the thorax, thus necessarily giving rise to a sound that has the pitch of vesicular breathing.

As we have seen, the cavities of the hemithoraces are practically independent of one another from the acoustic standpoint under normal conditions. In these two resonators, as a substitute for the air of the or-

dinary resonator, are placed lungs which, acoustically considered, are masses of innumerable multilocular cavities containing air.

Manifestly, if all of their parts are to be capable of perfect expansion, the lungs can not rest with their weight upon the floor of the cavities. They are, accordingly, held by virtue of the negative pressure—and so really by atmospheric pressure—in an intimate contact at all points with the walls of their containers. But while thus supported they are nowhere normally adherent to the cavity walls but glide freely without friction in adapting themselves to changes of volume. Little or no positive pressure is exerted during respiration upon the lungs. Positive pressure of any importance is only present under conditions that prevent respiration; that is, in straining while the glottis is closed. The purpose of these delicate adjustments is perfection of function in all parts of the lung, but it may be said that the lungs are also as perfectly adapted, from the anatomical point of view, to play the part of the air contained within a resonator as is possible for structures that contain solids. Furthermore, resonator action is not influenced in the case of respiratory sound by any of the provisions of nature which tend to restrict the dispersion of sound waves, for these sounds are, or may be, carried impartially to all parts of the lung.

Into resonators thus constituted are introduced through the bronchi, as it were through speaking tubes, a noise which originates during respiration in the vibrations of the vocal cords and which is composed of a variety of individual tones of low and of high pitch. It is a matter of observation that only the low-pitched tones penetrate freely the air-cells, the resonator tissue par excellence. Observation also shows that the pitch of the breath-sounds is the same as that of the percussion-note of the thorax, and is therefore of the pitch of the hemithorax as a resonator. In other words, since permitting the passage of vibrations which return as sound is equivalent to vibrating in response to these vibrations, we may say that the resonator vibrates to the introduced sounds, or, more correctly, that it exercises a selective action, emphasizing the tone in the noise which is of the rate of its proper vibrations, admitting other sounds, provided that they are low pitched, but, during inspiration—that is, during the period of its optimal tension—permitting barely a hint to be given of the presence of high-pitched tones. Vesicular breathing is due, then, to the action of the thorax-resonator upon laryngeal tones. Its specific character is not due to the laryngeal sounds in which it takes its origin, but to the selective and transforming action of the thorax-resonator upon the miscellaneous sounds submitted to it.

In the auscultation of respiration, as in percussion, we obtain evidences

of a local response with vibrations of the same frequency as that of the whole hemithorax. In quiet diaphragmatic breathing the sound of respiration is practically inaudible over the apices and, conversely, in apical breathing there is little or no sound over the lower lobes. In such localized respirations the sound of vesicular breathing is not, then, due to the action of the whole hemithorax and its contents, so far at least as the production of audible sound is concerned, but to the action of that portion of the respiratory apparatus which expands freely in inspiration and, therefore, has the optimal tension. In full respiration the breath-sounds are to be heard everywhere over the lungs. But the pitch of quiet breathing in a part of the lung and that of the full respiration of the entire lung is the same. Here again, therefore, as in percussion, the vibrations of a part equal the vibrations of the whole.

Since the laryngeal sounds are introduced into both lungs simultaneously and both halves of the thorax move in unison, it might be thought that in full respiration the whole thoracic cage functions as one resonator. The cavities of the hemithoraces are not, however, of exactly the same volume; at times the tone of vesicular breathing of one hemithorax seems to differ slightly from that of the other side and the sound of the upper lobes from that of the lower lobes of the same side. It seems more probable, therefore, that each hemithorax functions always in health as an independent resonator and possibly that one portion of the individual resonator may, at times, have a rate higher than other portions. In this latter case we might speak of the lung as forcing localized vibrations upon the chest wall, just as in percussion the chest wall forces localized vibrations upon the lung.

The resonator of the physicist is designed with a view to response only to its proper tone. Usually, however, while the proper tone is most strongly reproduced, there is some strengthening also of the neighboring tones which diminishes in both directions as the pitch of the tones recedes from that of the fundamental tone. This peculiarity which, according to Giesswein, varies in different resonators and is most marked in resonators with soft walls gives rise to the term "zone of resonance" (*Resonanzbreite*).[12] Besides the strengthening of the proper tone of the resonator and of its neighboring tones there is more or less strengthening, according to the nature of the tone-excitant and of the resonator, of the harmonic overtones, especially the octave. It may happen also that of two resonators of different shapes but adapted to the same tone one responds under certain conditions to a given tone while the other remains

[12] Giesswein, "Uber Brustresonanz," *Verhandl. d. deutsch. Gesellsch. Hals—, Nasen— u. Ohrenärzte*, 1921, 87.

silent. These differences are, as might be expected, particularly marked in attempts to analyze noises like those of percussion and respiration. It may be said, therefore, that only approximations to the scientific determination of the pitch of the thoracic sounds have as yet been reached by the use of resonators.

The studies of F. von Müller and his associates have added much to our knowledge of the laws of vibration of the thorax. Müller, investigating the pitch of the lung-sounds by means of specially constructed resonators, found that "the note of the normal lung elicited by percussion, normal respiration and the tones of a deep voice all correspond to a pitch in the large octave, or the beginning of the contra-octave. We are, therefore, justified in speaking of a fundamental tone of the lung which is deep because the inert mass vibrates slowly"; [13] and again: "Quite normal apices give only deep tones in inspiration but it is to be remarked that even in sound lungs the respiratory sounds of the apices generally furnish higher tones than those of the lower parts of the lungs." [14] While the chest is a resonator which responds strongly to its fundamental tone, there is a lesser response to tones of somewhat higher pitch. This is shown by the fact that, although a tuning fork of the same pitch as that of the dominant lung-note is heard best through the chest, forks of nearly the same pitch are heard well; the intensity of the sound, however, decreases as the pitch of the fork recedes from that of the fundamental lung-note. Testing the fremitus, it was found that the chest vibrates to the deep tones, but most strongly when the fundamental tone of the thorax was sounded. Selling and Müller, using their special resonators, found that the pitch of vesicular breathing lies in the highest notes of the contra-octave, extending into the large octave (60–80 vibrations per second). Gerhartz, according to Müller, has shown that the heart sounds have the same rate of vibration. The percussion-note is pitched with 54 vibrations.

Beginning with the left or bass end of the keyboard of the piano, the first C is the first note of the contra-octave. The octaves above the contra-octave are named respectively the large, small, one-lined, two-lined, three-lined and four-lined octaves. The "lined" octaves were formerly indicated by lines drawn beneath the letters that represent the notes. In modern usage accents replace the lines; figures may also be used, as a^3.

Selling found that while the normal note in his experiments was at A, in emphysema it was F and in pneumothorax E. Fremitus begins at f

[13] F. von Müller, "Zur physikalischen Diagnostik," *Verhandl. d. deutsch. Kong. f. innere Med., Wiesb,* 1911, XXVIII, 184.

[14] F. von Müller, "Diagnostik der Lungenkrankheiten," *Ztschr. f. arztl, Fortbild., Jena,* 1912, IX, 417.

and is loudest at E or F, the last tones representing the deepest tones of bass voices. The percussion-note of the young thorax is c, of children six years of age d to f. Bronchial breathing extends from the upper part of the one-lined octave into the two-lined octave. Pure bronchial breathing extends from d″ to d‴. The note of bronchophony lies in the one-lined octave. A sharp "ch" (German) has a pitch from b″ to g‴. Metallic tones, Müller says, must reach as high as 5000 vibrations per second. The variations in these figures are due, of course, to a considerable extent—at least in the lower-pitched tones—to the varying size of the thoraces examined. The figures are naturally only exactly correct in any case for the persons studied. For the sounds of low pitch they represent the lowest or fundamental tone. There are other tones present in sounds, most noticeably in musical sounds; the overtones or harmonics of the fundamental tone. As has been remarked, the sounds of respiration and percussion are "noises" made up of a great number of tones of which no single tone greatly predominates. Selling found that when a noiseless pleximeter (of soft rubber) was used resonators responded in an almost continuous series up to the two-lined octave. The higher pitched the tones, the weaker they were. With an ordinary ivory pleximeter, or with finger-finger percussion, a further series of high-pitched resonators responded (about to a″).[15] Gerhardt long ago analyzed the sounds of rapid and deep vesicular breathing with numerous small resonators and found that these sounds are composed of very many tones, all of them weak. But he probably did not use the tube that corresponds to the deepest tones, for which Müller found a length of two to three meters to be necessary.

As respects the vesicular murmur, the thorax is analogous to the flue pipe of the organ. Here the air-current of normal respiration, striking against the slightly projecting vocal cords, produces a noise made up of a variety of tones. Among these is one or more capable of exciting the fundamental tone of the thorax. Without such an excitant the thorax is silent. It is true that the phenomena of reciprocating breathing show that sound may be produced within the lung by the motion of air. This is necessarily to be expected from the general laws of the formation of sound in tubes. It is all a question of the amount of energy expended. In reciprocating breathing the air concerned in the production of sound is under considerable positive pressure. It is forced powerfully through certain bronchi by the abnormally strong action of localized portions of the respiratory apparatus, whereas in normal inspiration there is no positive pressure. The air, on the contrary, is driven in from without

[15] Selling, *loc. cit.*

to equalize a commencing rarefaction. From the nature of the case the pressure within the lung, in normal quiet inspiration, can not exceed that of the atmosphere without. And the force of the air-current, in reciprocating breathing, must be immensely greater than that of the strongest normal inspiration.

The thorax as a resonator is unable to respond to the higher-pitched tones contained in the laryngeal noise of normal respiration, in which respect it resembles other resonators. In this case the sounds are too weak to force themselves upon the resonator. But if the breathing is increased in force and becomes harsh, somewhat higher-pitched tones are transmitted through the thorax to some extent.

As respects vocal resonance and fremitus, the thorax resembles the reed pipe of the organ. For, in phonation, a series of sound waves of definite pitch are produced by the action of the vocal cords and their vibrations, conducted by the bronchi as speaking tubes and force themselves on the thorax, so that it transmits and can be felt to vibrate to the tones of the voice. But, as in the case of the reed pipe, only those vibrations which are not widely different in their periodicity from that of the thorax can produce the full vibrations of the chest wall that show themselves as fremitus; high-pitched sounds are not transmitted at all.

The Resonators of the Human Body Physiologically Considered

This chapter is not complete without some reference to the relation of the resonators of the human body to its physiological activities.

It is of no advantage to the organism that the thorax is a resonator; its properties as such are the inevitable result of the general laws of sound. The middle ear is the only resonator of which the acoustic properties are of prime importance. The middle ear is as inevitably a resonator, in the sense of having a fundamental tone, as the thorax. But, evidently, for the perfect working of an organ, the function of which is to transmit impartially all sound, resonator action is precisely the property that must be most sedulously avoided. It is minimized, accordingly, by the smallness of the cavity, which results in a high pitch of the fundamental tone and a rapidity of vibrations of its overtones that places all or most of them above the range of audibility; the irregularity of shape and the thickness of its walls prevent too ready response to any single tone; the mucous membrane which lines the cavity of the tympanum absorbs sound and its softness makes impossible the rapid superficial vibrations which might give rise to the audible high-pitched overtones

of relatively low tones that are forced upon the ear. These provisions are all-important, for over the cavity is stretched a membrane which, without them, would make the air-space beneath a resonance-chamber to reinforce and thus pervert the true nature of external sound. The membrana tympani is a wonderfully impartial transmitter of sound, but even a stretched membrane has periodic vibrations; these are, however, much less obtrusive than the vibrations of an air-containing cavity. Difficulties of this kind, which are inevitable, are avoided largely by the necessarily high pitch of the objectionable sounds. The sounds described as "ear-piercing" are of high pitch and probably approximate to the fundamental tone of the tympanic cavity. According to Helmholtz the sounds especially intensified in the human ear are those that lie between e'''' and g'''' and embrace between 2640 and 3168 vibrations per second (this assumes 264 vibrations for middle C). This explains the peculiarly sharp, cutting effect of certain notes in the neighborhood of f''''. The chirp of the cricket is grating because so nearly of the proper tone of the middle ear.

In the thorax, on the other hand, the evil effects of resonance are minimized by the low pitch of the fundamental tone. Certain men, however, according to Selling, experience disagreeable sensations when deep organ notes are sounded, probably because the pitch of the fundamental tone of their thoraces is reached or approximated.

The Transmission of High-Pitched Sounds Through the Thorax

The transmission of high-pitched sounds through the thorax presents a problem which will be comprehended more easily after a brief consideration of the transmission of similar sounds which do not originate within the human body.

If one listens to the ticking of a watch, besides the dull, medium-pitched sounds of the watch movement one hears in all but the cheapest grade of watches a very high-pitched and faint metallic ringing sound, but only when the watch is pressed against the ear. Although the lower-pitched sounds remain perfectly distinct to a considerable distance, the high-pitched sounds disappear as soon as the watch is in the least removed from the ear. When the watch is laid upon a table the high sound can be heard very imperfectly at a distance of an inch or two, if the ear is pressed very firmly upon the table, but the other sounds of the watch are heard with great distinctness over the full length of the table and could evidently be heard for a much greater distance. The high-

pitched sound is not heard through the bell of a binaural stethoscope pressed against the face of the watch, but is very clearly audible through a wooden cylinder used as a monaural stethoscope and applied to the watch in the same way. These observations show that the high-pitched sound is well propagated through elastic solids provided that there is no break in the acoustic medium. Lying upon the table the watch was not in close contact with its surface and conduction was poor for auscultation through the wood of the table top, but became good when firm pressure gave close contact of the wood of the cylinder with the watch crystal. But the high-pitched sound of the watch does not appear to pass through the air at all.

A silver coin pressed against the tragus and percussed with the edge of another coin gives a dull sound from the full contact of the two surfaces mingled with a high and not loud sound from the vibrations of the superficial particles of the coins which are directly implicated by the blow. Listening in this way one gets the full strength of the silvery sound. If the coin is laid upon the table and percussed in the same way the silvery sound does not reach the ear through the air nor is it transmitted to the ear pressed against the table. This sound is not transmitted through healthy lung but is heard very distinctly in most cases of pneumothorax, when it has a more bell-like quality due to the resonance of the cavity. If the coin is placed over the region of absolute heart-dullness and is percussed by an assistant, when auscultation is performed posteriorly from above downward, a dull, "wooden" tone is alone heard so long as one is over the lung. According to Jacobson and Danielopolu at the level of the tenth dorsal vertebra the sound becomes silvery during expiration and wooden again on full inspiration, showing the effect of the intrusion of the lung. At the twelfth vertebra the sound is silvery; farther down it becomes Trousseau's "copper" sound which is normal for the abdomen.[16] We conclude from these observations that the silvery sound—like the high-pitched sound of the watch—is well conducted through solid media, in this case the liver.

The Metallic Sounds

The percussion-sound, the breath-sounds and râles may all have a metallic quality which is due to the presence in the sound of high-pitched tones similar to the sounds that have just been mentioned. It is essential for the conduction of these sounds that they shall not pass through the air-

[16] Jacobson and Danielopolu, *München. med. Wochenschr.*, 1912, LIX, 216.

cells. They are produced in a cavity with firm and elastic walls. In order that the metallic sounds of respiration may originate in a cavity it is necessary that it have a shape such that entering air-currents pass directly into the air of the cavity without impinging against its walls. That is, the shape of the upper surface, or roof, of the cavity should be something like the top of a large bottle with a narrow neck (the latter representing the bronchus). Air entering a cavity in such a way is said to be broken up into quite irregular waves. The sound waves resulting from all of these vibrations would combine to make a noise made up of many unrelated tones. But of these vibrations only those that are capable of exciting the fundamental tone of the cavity experience full reinforcement. If the current of air is forcible or if the conditions are otherwise peculiarly favorable, the agitation of the air may result in producing also a series of very high-pitched vibrations, the metallic breath-sounds. It is found that metallic percussion-sounds are best elicited by percussing with a hard and not heavy object (like a pencil) upon a metal pleximeter. In such percussion, tones of a high frequency are produced, but these are damped by the sluggishly vibrating normal lung and remain inaudible. But if a large, empty or nearly empty cavity with firm walls lies beneath the point of percussion, such high-pitched sounds may correspond to the high overtones of the fundamental tone of the cavity, in which case they are heard as metallic sounds; for a resonator may show specific resonance, though feebly, to the overtones of the fundamental tone. (See p. 78.)

The bursting of bubbles through a tough mucus in such a cavity may give rise to râles of a metallic quality. The relatively slow current of air in respiration causes more often the fundamental tone of the cavity, the amphoric echo, than the metallic sounds. If the latter are heard the amphoric echo is usually also present and the same is true of the metallic râles. In percussion with a light object like a pencil, however, the percussing mass is so small that it does not bring out strongly the deep tones and the high-pitched sounds may predominate, the amphoric echo being faint or inaudible. The point that should be emphasized is this: the metallic sounds, however produced, are always the high disharmonious overtones of the fundamental tone of the cavity. We may say, then, that the metallic sounds experience a reinforcement as truly as the fundamental tone. There may seem to be a difference of behavior in the metallic sounds between the metallic sounds produced outside the body and those that pass through the thorax. The former do not survive a change of sound medium—they are not conducted through the air at all—the latter, even when produced in the air of the cavity—though they

are obliged to pass from air to solids in their transmission through the chest wall—are easily to be heard by the ear applied to it. Again, there is an apparent inconsistency in the behavior of the silvery sound of coin-percussion. This sound, as we have seen, is heard better in pneumothorax than when the coin percussed rests directly upon the ear, but is inaudible over the healthy lung. Yet it is well conducted by the chest wall in health at a level below that of the lung. There might seem at first sight to be no reason why the silvery sound of percussion, like the ordinary sounds of percussion, could not be conducted around the normal chest in the tissues of its wall without the need of passing through the lung. Both difficulties are solved when we remember that here we have to do with resonators. In the first case the cavity of the pneumothorax, acting as a resonator, reinforces the metallic sounds as overtones of its fundamental tone and, since the thoracic wall vibrates as a part of the resonator, the sound is heard by the ear, as it were, at the very place of its production. In the case of the coin-test in pneumothorax, as in that of the watch sounds, the high-pitched sounds are really carried from a solid medium directly to the ear. But when the coin-test is applied to the healthy chest the thorax is the wall of practically a different resonator—one filled with lung that vibrates slowly. The chest wall is abundantly able in itself to vibrate at the rate required to transmit the coin sound, but, as a resonator, it is governed by the periodicity of the vibrations of its contents; like the glass that contains a foamy liquid, it cannot vibrate independently while it contains the expanded lung and the lung is not capable of a sufficient rapidity of vibration to transmit so high-pitched a sound. In other words, it damps the high-pitched tones in that part of the thoracic walls with which it is in contact, but is unable to prevent their transmission below its level.

THE EXAMINATION OF THE HEALTHY CHEST

It is obvious that one cannot be competent to decide as to the abnormality of the conditions found by auscultation and percussion of the chest, unless he knows what the signs of the healthy chest are and how they are influenced locally by peculiarities of the normal anatomical structure of the parts. Yet the study of the normal chest has been altogether too much neglected in the past and, as the result of this neglect, some important and easily ascertainable facts have been ignored or misinterpreted.

The Conduction of Sound by Bones.—In a frozen section through the posterior mediastinum it is seen that the spine projects nearly to the

middle of the thorax and has in front of it the aorta, the esophagus and the ventricle, yet it is known that percussion of the lower dorsal vertebrae gives, not dull or weak sound, but a loud, deep lung-note which only passes into a weak note at the diaphragm. This can only be explained by supposing that the lung beside the vertebral column resounds when the spine is percussed. And, as F. von Müller [17] from whom the foregoing sentence is quoted points out, if the percussion had acted only in a sagittal direction a weak sound must have resulted. ,Bones manifestly have the property of conducting a blow, not only in the direction of the stroke but in all directions. And they conduct equally well sounds that originate within the body. The breath-sounds of the trachea are well conducted to the acromial end of the clavicle in most persons. Erdman [18] has recommended auscultation at this point in the diagnosis of fracture of the clavicle with the idea that a fracture in that bone would cause an interruption in the propagation of the sounds of breathing. He found that in ninety-five per cent of healthy persons a more or less intensely bronchial whisper could be heard over the acromial end of the clavicle. Similarly, breath-sounds are conducted from the pharynx through the bones of the head and may be heard distinctly over the temples and occiput, where they have a marked bronchial or tracheal quality. Transmission of the whisper to the lower cervical and upper dorsal vertebrae is a normal phenomenon.

The lung-resonance is communicated so freely to the sternum that percussion upon the manubrium gives but slight, if any, change in the note from that over the lung at either side of the bone; the same is true of auscultation over the sternum. Conduction of sound by the ribs has already been referred to in the chapter upon Percussion.

Plesch [19] has devised a method of immediate percussion of the upper chest which is frankly calculated to elicit the resonance of the apex as a whole. It is mentioned in this connection because it is an interesting acoustic experiment rather than a contribution of value to practical diagnosis. Percuss lightly with the middle finger of the right hand the incisura semilunaris sterni (the upper edge of the manubrium). Press with the four finger tips of the left hand, not very strongly, at the middle third of the intercostal space immediately below the clavicle upon either side. This pressure damps the vibrations of the apex of that side so that the resonance of the blow upon the sternum is communicated chiefly to the other side. Noting the sound produced, the observer then damps in

[17] *Ztschr. f. ärztl. Fortbild., Jena,* 1912, IX, 417.
[18] *J. Am. M. Ass., Chicago,* 1911, LVI, 740.
[19] *Deutsche med. Wochenschr., Berl & Leipz.,* 1911, XXXVII 825.

the same way the apex of the other side and the resonance which results is compared with that of the side first percussed. It is of course important that the blows shall be of equal strength during the examination of the two sides.

The connection of the first rib with the sternum is usually by bone; the second and lower ribs, on the other hand, are connected by articulations. The result of the anatomical structure is that vibrations excited in the upper part of the sternum are transmitted much more forcibly to the apex than to the lower parts of the upper lobe. This method has the advantage that, since the same place is percussed for both apices, changes due to varying thickness and tension of the muscles and to variations in the application of the pleximeter are excluded and that the blow of percussion should excite an equal amount of sternal resonance whichever apex is under examination; so that the resonance of the two apices should be strictly comparable the one with the other.

The curved shape of the ribs diminishes their power to vibrate when percussed. This loss of capacity for vibration is the more marked the sharper the curvature. At the angle of the ribs where the bend is sharp there is normally dullness on percussion. Deformities of a minor sort due to rickets, pigeon-breast, funnel-shaped depressions of the sternum, or symmetrical protrusions of the lower ribs that are attached to the sternum near their junction with that bone have a considerable effect upon the percussion-note. Still more important are the thoracic malformations due to scoliosis. The lateral deviation of the spine is always accompanied by torsion of the vertebrae; the shoulder is higher and the back protrudes more on the side of the convexity of the curve—the scoliosis is really a kyphoscoliosis. Each pair of thoracic ribs may be conceived of as forming a hoop the halves of which are connected by sternum and spine. Since the torsion affects the entire circumference of the hoop, its greater bending in the back is accompanied by a similar, less-marked bending on the opposite side of the body in front and by a corresponding flattening through the remainder of the course of the ribs. Increase of convexity of the rib tends to lessen, flattening of the rib tends to increase its vibratility. We shall have, therefore, dullness on percussion posteriorly on the side of the convexity of the scoliosis over the hump (that is, the region in which the ribs are abnormally convex) and a lesser dullness anteriorly on the side of the concavity over the anterior hump, while the resonance of the side of the concavity posteriorly and of the side of the convexity anteriorly, so far as the ribs influence the percussion-note, will be good. If we suppose the convexity of the scoliosis to be to the left, as

it is in the majority of cases, there would be dullness over the left chest posteriorly and a lesser dullness over the right chest anteriorly (in the region of the costal cartilages), and over the right chest behind and the left chest in front the resonance would be unimpaired.

But the effect of the scoliosis upon the chest is not limited to its own level. There is a compensatory curvature of the spine below the scoliosis in the opposite direction. The hoops are united to form a barrel—the thoracic cage. A twisting and bending of one part of the barrel must result in a compensatory twist and bend in the opposite direction of the hoops of the other part. The percussion findings will be correspondingly reversed; that is, the left back, dull above, is resonant below; the right back, resonant above, is dull below; in front, the right side, dull above, is resonant below; finally, the left side, resonant above, is dull below. There are then four regions of dullness in scoliosis: 1. Above, on the side of the convexity posteriorly; 2. Above, on the side of the concavity anteriorly; 3. Below, on the side of the concavity, posteriorly; 4. Below, on the side of the convexity anteriorly. In these designations of locality the primary superior convexity and concavity are of course meant. It is to be noted that the findings in the lower series are reversed for the reason that the convexity and concavity of the secondary deviations are reversed. The dullness behind at the base, for example, is situated in the region of secondary convexity, just as above it is in the region of primary convexity. The degree of dullness diminishes progressively from one to four in the above series. Neumann [20] has known the dullness of the kyphoscoliotic region, both anterior and posterior, to be interpreted as indicating tuberculosis and the dullness of the base behind to be regarded as of pleuritic origin, but has never known a mistake to be made because of the presence of the slightly marked fourth region of dullness. As a rule the effect of the secondary deviation below is so slight as not to be readily detected. It is well to examine as many cases of minor thoracic deformity in individuals free of the suspicion of tuberculosis or other lung disease, as possible, in order to become familiar with the effect produced by malformations upon the percussion-note, before attempting to make allowance for the share that deviations from the normal in the skeleton may have in affecting the signs of pulmonary tuberculosis.

When the back is strongly bent its arching results in a deadening of the resonance of the thorax. It is the low-pitched tones that are sup-

[20] Neumann, "Lungenbefund bei Skoliosis," *Beitr. z. Klin. d. Tuberk., Würzb.*, XVIII, 399.

pressed. The percussion-note may even be louder when the back is arched, but in this case the pitch is noticeably raised. The subject should only bend slightly forward when the back is percussed.

The Thorax and the Lungs.—There are two extreme types of chests: the short and thick chest of the stockily built individual and the long and narrow chest of those who are less well developed. The chests of most persons fall somewhere between these two extremes, partaking more or less distinctly of the peculiarities of the one or of the other type. The importance of this distinction of types is this, that many signs which would be abnormal in the deep chest are not necessarily abnormal in certain portions of the thorax that is long and narrow. Whether the sounds of the tubes shall succeed in penetrating through the mass of air-cells of the lung depends upon the depth of that mass, which will be the less at any given point on a horizontal plane in the long-stretched chest than in one that is short and thick.

The characteristic note of the air-cells is best obtained, both in auscultation and in percussion, over the lower lobes at a level below that of the angle of the scapula. For in the lower lobes the bronchi are rapidly reduced to tubes of small calibre and approach the surface of the lung in a direction perpendicular to that surface. There is, therefore, here a minimum of bronchial sound because the mass of the air-cells is the greatest and the size of the more superficial air-tubes the least of any part of the lung. In the upper lobes, on the other hand, the mass of air-cells is smaller and the large tubes approach more closely to the surface of the lung and in some locations run approximately parallel to the chest wall. The bifurcation of the trachea is situated about on the level of the third rib at its junction with the sternum; the main bronchi and the trachea are here not deeply covered with lung and furnish an important element in the lung percussion-note, the more prominent the longer and narrower the chest. We find on careful percussion that the note of the lung above the third rib has a shorter and less resonant sound in the long and narrow chest as compared with the note lower down and that the breath-sounds likewise are of a slightly sharper quality. In the apices the percussion-note and the breath-sounds show the same characters to a marked degree and this is especially noticeable on the right side on account, it is thought, of the proximity of the trachea chiefly; but the difference between the two apices and the change in the quality of their resonance are not always apparent in the thick and short chest. The question of the normal signs of the right apex is so confused by the possibilities of obsolete morbid changes in that region that further con-

sideration of the apex will be deferred until we come to speak of the physical signs of disease.

As Fetterolf has shown, the trachea lies almost entirely throughout its thoracic course in contact with the right lung, separated from it only by the parietal pleura and a delicate layer of areolar and lymphatic tissue, while on the left side the lung is separated from the trachea by large blood-vessels, by the esophagus and by areolar and lymphatic tissue to a depth of about 3 cm.[21] The result of this closer contact on the right side is better transmission of sound from the air-tubes; this shows itself by slightly harsh breathing and slightly prolonged expiration which are most marked at a point in the second intercostal space close to the sternum. From this point the change of sound continues to be apparent with gradually diminishing intensity over the second intercostal space for the distance of two stethoscope bells, more or less, and is also perceptible in the third intercostal space near the sternum. Vocal resonance and fremitus are likewise increased over this area. There is much difference in different individuals in the degree to which the sounds are altered and the extent of surface over which the change is to be made out. These sounds should be studied carefully in the healthy subject in order that they may not be confounded with signs of disease.

Increased vocal resonance and slightly harsh breathing which do not appear to indicate disease are sometimes to be detected immediately below the center of the left clavicle. Slightly prolonged expiration at the left base posteriorly is sometimes heard. It is said to be normal, but is difficult to account for at that place. Very slight harshness of respiratory sounds with prolonged expiration in the lower paravertebral regions of both lungs posteriorly, most marked at about the angle of the scapula, disappearing a short distance above that point, equal on both sides, or slightly more marked at the angle on one side—more frequently the left—are due to the proximity of the bronchi which supply the posterior portions of the lower lobes. Like the other changes which depend upon increase in the bronchial element in percussion and auscultation, they are more manifest in the long and narrow chest. The changes are very slight; only to be made out by the closest observation and concentration of the attention. They are of course to be distinguished from the bronchovesicular quality often apparent in the vicinity of the hilus higher up, which is also thought to be due to the nearness of bronchi to the surface. The changes now under consideration are not often likely to prove misleading, but should be studied for the reason that ability to detect them is an excellent test of the proficiency of the observer in physical diag-

[21] *Arch. Int. Med., Chicago,* 1909, III, 25.

nosis. Some physicians who are pretty good diagnosticians fail to make them out when undoubtedly present. It is not known, of course, that they are invariably present in the short thick chest, but they are found in many men of athletic build and failure to detect them, in the long and narrow chest at least, is evidence that the student has still something to learn.

In Traube's semilunar space the percussion-note has a mixture of the tympanitic quality with the normal pulmonary resonance. This space is bounded above by the unmixed pulmonary resonance and below by the tympanitic resonance of the intestine. The tympanitic component of the percussion-note, being due to the proximity of the stomach, is of lower pitch than that of the smaller cavity of the intestine but is higher-pitched than the pulmonary resonance. The boundaries of this space are, of course, not sharply marked. Percussion of this region should be practiced as an exercise in the study of the tympanitic percussion-note.

Sternal Sounds; Sounds in the Chondrosternal and Other Joints; the Scapula Sound; Marginal Sounds, Sounds at the Lingula.—We now proceed to consider a class of sounds which simulate more or less perfectly the râle and which when taken for signs of disease have led to innumerable serious errors of diagnosis. They should by all means be studied with care upon the healthy subject.

Sternal Sounds.—Fine sounds resembling crepitant râles, but not quite so fine and dry, are heard during deep and forcible inspiration over the sternum. The quality of the sound is different from that of pulmonary râles; the word "cartilaginous" best describes them. The crepitations are numerous; there is a shower of fine crackles. They are sometimes heard over the manubrium, sometimes only in the vicinity of the ensiform appendix, but are usually heard best over the gladiolus. They are sometimes confined to a limited area in ordinary breathing, while inspiration, following expiration and cough, develops them over the entire sternum. On account of their fineness these sounds are not as a rule transmitted beyond the sternum. They may be elicited near the insertion of the great pectoral muscle when the arm is swung forcibly from before backward, the breath being held. They are believed to be due to the bending of the bone by the respiratory act or by the tugging of the attached muscles. They may be produced by bending a freshly removed sternum.

Sounds in the Chondrosternal Articulations.—These sounds are heard loudest over the chondrosternal articulations during deep inspiration. They imitate medium-sized moist râles, from which they are distinguished not only by a slight difference in quality but also by the fact

that they are loudest over the joint and may be transmitted, with diminishing intensity, along the ribs. They are also transmitted to the neighboring soft parts but do not appear to be propagated to the other side of the chest. These sounds are sometimes brought out by swinging the arms while the breath is held. In the writer's experience they are heard most frequently over the second costal articulation of the left side. Though usually confined to one joint, in one case they were heard over the second, third and fourth articulations of the left side at the same examination and were propagated along the second rib to the mamillary line and nearly to this line along the third rib, the subject being a young and athletic man in good health. Similar sounds may be heard on auscultation of a sternum while an assistant holds the bone firmly with one hand and moves the attached costal cartilages with the other. Less frequently, the sounds originating within these articulations are clicks resembling more closely ordinary joint sounds. They are then single sounds, rather coarse, of a "bony" quality, not usually loud, are propagated along the rib to which the articulation belongs and may occasionally be transmitted through the sternum to the other side of the chest. The clicks are comparatively rare and have only been found in men of athletic training. It appears probable that they are made possible by the stretching of the joint capsules in the athlete which permits a greater separation of the articular surfaces than is found in persons whose thoraces have not been subjected to severe strains. All classes of sternal sounds are best produced during the inspiration that follows expiration and cough and the frequency with which they are found will depend largely upon the proficiency that the subject has attained in this method.[22]

The sternum is usually auscultated for the determination of increased conduction of sound due to the presence of tumors or other morbid conditions of the anterior mediastinum. Perez [23] found in 1895 that elevating or lowering the arm produced "crepitating friction sounds and crackles over the sternal region of a tuberculous nature" in a patient with a tuberculous history who had suffered from what had been diagnosticated as dry pericarditis or mediastinitis. W. Ewart [24] examined this patient and suggested that these sounds were produced in the joints that were subjected to motion, stating that he had heard the same sounds in the shoulder joint. But he was evidently not entirely satisfied with his interpretation for he goes on to suggest that "shoulder-girdle conduction" is capable of

[22] G. E. Bushnell, "Extrapulmonary Sounds which Simulate Râles," *Med. Rec. N. Y.*, Jan. 20, 1912, LXXXI.

[23] Geo. V. Perez, *Brit. M. J., Lond.*, 1896, I, 82.

[24] Wm. Ewart, *Idem*, 1912, I, p. 771.

picking up audible vibrations of extra-articular origin—in other words that the sounds heard in the shoulder joint may have originated in the sternal region—and remarks that "it is high time that the analytic method be applied for the elucidation of the most difficult group in the entire series (of sounds of the upper thorax), the study and identification of those intrathoracic motor crackles which were first introduced to our notice by Perez." The discovery of Perez was made in a patient whose attacks of anginal pain had directed attention to the sternal region and it was natural that in a patient with a tuberculous history the sounds should be thought to bear some relation to a tuberculous process, as it may be that they did in that particular case.

However, in view of the frequency of these phenomena in the healthy individual it is going too far to say with some authors that "the only positive sign of acute mediastinitis is the presence of a mediastinal crepitation, or a quasi-friction sound elicited by deep respiration," or that "a creaking or other sound audible on auscultation when the patient moves his arm may give definite evidence of the presence of chronic mediastinitis."

This point is dwelt upon in order to emphasize again the importance of making sure that a physical sign does not occur in health before declaring it to be a sign of disease.

Evidently also if the finer sternal sounds should be interpreted as pulmonary râles the mistake might lead to serious errors in diagnosis. When the sounds in the chondrosternal joints resemble the coarser and moister râles the distinction is difficult and in several instances known to the writer these sounds have led to the incorrect diagnosis of pulmonary tuberculosis. In one case an army officer, whose health is always conspicuously good, stated that his chondrosternal sounds (which imitate râles most deceptively) are invariably at first regarded as evidence of a tuberculous lesion whenever his chest is examined. The clicks, with proper attention to their quality, should not be mistaken for intrapulmonary sounds. To show that sounds heard in this region are not sternal sounds it is imperative that the subject be required to swing his arms or move his shoulders backward and forward vigorously, the breath being held during this movement. The procedure sometimes develops an astonishing number of sounds that resemble moist râles.

The movements of respiration or the varying tension of muscles may produce sounds in the shoulder joint and in the articulation of the clavicle with the sternum. Sometimes these sounds are coarse and with a little attention should not be confused with intra pulmonary sounds. In other cases they have more of a rumbling or grating character and may be mis-

taken for friction sounds. Change of position often causes them to disappear. They may persist when the breath is held and the shoulder is moved.

Scapula Sounds.—In certain positions of the body, particularly when the body is somewhat inclined forward and the shoulder advanced, the scapula knocks against the ribs and produces a rather loud sound which may be heard by the bystanders. The production of this sound is much facilitated by emaciation, consequently it is common in consumptives. It has, however, of course no direct connection with tuberculosis.

Muscle Sounds.—Sahli divides muscle sounds into active and passive. The active occur in shivering, from fibrillary contractions that cause audible sound long before visible shivering is present. Respiratory contractions are, however, the main cause. A spasmodic contraction of bundles of muscle-fibres produces a dull, low-pitched and more or less continuous sound that resembles friction-sounds rather than râles. It is caused to disappear by inducing the patient to relax his muscles which he is holding unnecessarily tense.

Intermittent contractions of muscle-fibres may cause sounds that closely resemble "dry" râles and give rise to much confusion. They are particularly common in the trapezius but Webb has noted them in contractions of the platysma.[25] In the examination of more than 20,000 men at Chickamauga a record was kept of 819 examinations for adventitious sounds over the upper lobes of the lungs. According to King,[26] for each man found to have tuberculosis there will be more than two who will have crackles of no significance. King found that sometimes the sounds were heard distinctly when the stethoscope was directed inward to the neck muscles above the clavicle, similar sounds of less intensity being heard at the same time over the lung-tissue. They are produced, in his opinion, when the subject is in the position customary in examinations—with the head turned away from the side that is being examined. Sometimes the sounds disappeared when the face was turned forward or slightly toward the side examined, but the most helpful procedure was to cause the subject to extend the head backward upon the neck as far as possible, which position stretches the neck muscles. In the writer's experience sounds of this kind have rarely caused difficulties, probably because he does not require his patients to turn their heads away during examination.

Sometimes bundles of muscular fibres slip under the pressure of the stethoscope and produce sounds that somewhat resemble râles. This is

[25] G. B. Webb, Personal Communication.

[26] John T. King, Jr., "Auscultation of Pulmonary Apices in Young Men," *Mil. Surgeon.*, Jan., 1918.

particularly likely to occur over the anterior fold of the axilla. These are passive muscle sounds.

In estimating the significance of sounds resembling râles heard above the clavicle the location of the upper boundary of the lung should be borne in mind. The apex of the lung rises above the clavicle in close proximity to the neck and is accessible to percussion between the sternal and clavicular heads of the sternocleidomastoid muscle. Outward from the apex the supraclavicular fossa and the parts behind and above extending to the top of the shoulder, are comparatively distant from the lung the resonance of which is, however, obtained over Krönig's isthmus, being conveyed upward from portions of the lung situated behind and below the clavicle. Hence percussion directly backward in the middle of the supraclavicular fossa reaches the lung only in an indirect way. The same is true of auscultation. Breath-sounds in the supraclavicular fossa

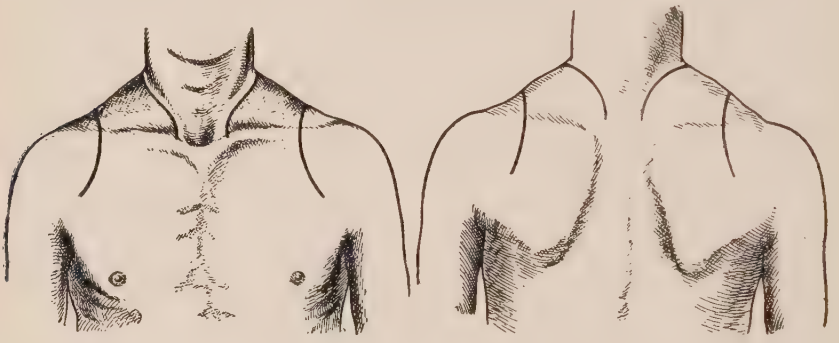


Fig. 16.—Anterior resonant area over normal lung apices.
Fig. 17.—Posterior resonant area over normal lung apices. (Krönig, Deutsche Klinik, Berl. & Wien, XI.)

are conveyed upward from the more deeply situated lung to the muscles which bound the fossa posteriorly. It follows from these facts that sounds which resemble râles in the supraclavicular fossa, if they are really pulmonary râles, are conveyed either from the true apex or from the edge of the upper lobe. In the first case these râles should be heard in the true apex; in the second they should be heard below the clavicle. If they are not heard in either of these places they are probably not râles but muscle sounds. Furthermore, since the upper edge of the lung in its extension downward and outward from the apex is relatively thin, any tuberculosis focus situated in it might be expected to be accessible to auscultation and percussion posteriorly. If there are no râles to be heard behind over the upper edge of the lung, the probability is increased that sounds heard in the supraclavicular fossa are not true râles.

Krönig described in 1889 a method which he called topographical percussion of the apices.[27] The examiner, standing at the side of the patient, begins at the root of the neck on the top of the shoulder and percusses out toward the shoulder joint until he obtains pulmonary resonance; marking the point, he then continues the percussion at other points in front of and behind the first position until the points of first resonance, joined in a line, indicate the mesial boundary of pulmonary resonance. In the same way the outer boundary of pulmonary resonance is outlined. The result is the marking of an area narrow on the top of the shoulder and broadening in both directions as it descends. At the narrowest part which, after Wolff-Eisner, is called the isthmus, the breadth of the resonant area is 4–5 centimetres in the normal chest. The boundaries are quite distinct in the normal lung. If an active tuberculous process is present the lines become "washed," it is impossible to determine the exact line of demarcation, but in old fibrous lesions the line becomes even sharper than in health. The percussion is always performed on both sides so that the relative extent of the lung-resonance in each can be compared. Since the top of the shoulder is not in close contact with the lung, it is surprising that the pulmonary resonance should be so marked as it is. The sound obtained by percussion is the projection upwards of the resonance of the upper lobe. Why under the circumstances the lines of demarcation should be so sharp has not been explained. The value of the method has been disputed by some, but the reason of the failure to appreciate it is that the object sought has been chiefly the early diagnosis of active lesions. It is true that, as has been remarked, the lines become blurred when there is underlying congestion but recent tubercule may not be disclosed at all by the percussion, or be shown by such slight changes in the percussion-note as to be unreliable as a means of diagnosis. On the other hand, the method is valuable in the detection of retraction of the upper lung, furnishing a permanent record of the shrinking that results from a chronic tuberculous process. If there is bilateral disease the narrower isthmus shows on which side the lesion has most retracted—therefore, usually, the older lesion. The true utility of the method consists in the determination of the width of the isthmus; the percussion by Krönig's method of the lower portions of the resonant field, where lung-tissue is directly subjacent, offers no advantage over the ordinary methods. (See p. 22.)

Quiet expiration is effected by the relaxation of the muscles used in the preceding inspiration which permits the return of the chest to its position of rest through the elastic recoil of the ribs and of the abdominal

[27] G. Krönig," Zur Topographie der Lungenspitzen u. ihre Perkussion," *Berl. Klin. Wochenschr.*, 1889, No. 37. Also, *Deutsche Klinik, Berl & Wien,* XI, 581.

walls. It is, therefore, a passive process. The untrained subject when asked to breathe very deeply naturally fixes his attention solely upon inspiration. He takes deep breaths and throws out his chest with the idea of making it as large as possible, but gives no thought to expiration. The result is that, if this mode of breathing is long continued, the lung, never being efficiently emptied, becomes overfull and a stage is reached when the subject is unable to take in any more air and is obliged to ask for a rest. Here the limits of respiration have been for expiration the position of rest and for inspiration fullest inflation. The unopposed exertion of the respiratory muscles has overfilled the lung which, not being a perfectly elastic organ, requires some time to return spontaneously to its former volume after distention. Expiration, though passive in quiet breathing, is necessarily an active process when the work performed is severe and rapid; the individual who breathed so poorly under examination, if he had been required to run rapidly and if his condition was at all adequate to the exertion, would have made use instinctively of the accessory muscles of expiration. His breathing would have become panting respiration. Any one who observes a panting man or dog can not fail to note that the stress of the respiratory efforts is laid upon expiration. When after severe exertion of this kind the individual "gets his breath," not only has the heart become quiet and the congestion of the lungs abated, but the panting breaths in which expiration is more forcible and prolonged than inspiration, continuing some time after the exercise has been discontinued, have gradually reduced the distended lung to its normal size. Since breathing usually requires active muscular contraction in inspiration, it may be said that every one knows how to inhale. Improved control of the respiratory act lies almost altogether in the direction of a better regulation of expiration.

The contraction of the accessory muscles of expiration diminishes the size of the abdomen and raises the abdominal pressure, crowding the viscera upward and thus pushing the diaphragm above its position of rest. Another type of respiration is therefore possible, that in which the range is between the position of rest and that of active expiration. Starting to exhale from the position of rest, the subject expels the air with the aid of the accessory muscles of expiration. The lower part of the thorax, which has been drawn inward beyond its position of rest, expands in the following inspiration by virtue of its elasticity, the muscles relax and the viscera descend to their position of rest, carrying with them the diaphragm. To a certain extent, therefore, and especially in the erect position, inspiration becomes a passive process. We have then three extreme types of respiration: first, expiration passive, inspiration active;

range, from inspiratory expansion to the state of rest; second, expiration active, inspiration passive; range, from state of rest to that of expiratory contraction; third, deepest respiration in which respiration and expiration are both active processes and the range is between fullest inspiratory expansion and greatest expiratory contraction.

The position of rest may be defined as that in which no muscular contractions are in operation either to increase or to diminish the volume of the lungs. It is reached when the elastic recoil has expended itself and the muscles have not begun to contract for the succeeding inspiration. Normal breathing passes from the first type to the third as the respirations increase in force. It is to be noted that no mode of breathing can be long continued unless the intake and outgo of air are equal; if inspiration exceeds expiration in volume the lung becomes overfull and if expiration should long exceed inspiration the diaphragm would be pushed up to an unwonted height, the lung would be almost deprived of air and, before long, grave respiratory and circulatory disturbances would ensue. On the other hand, any type of breathing may be continued indefinitely without unduly expanding or contracting the lung, provided that the effective force of inspiration and of expiration are equal.

The practical importance of the second type of breathing is to be found in its use in the method known as the method of "expiration and cough." This method, with which every student of physical diagnosis should be familiar, has for its chief object the detection of moisture within the lung. It is best practised upon the healthy subject. Starting with the position of rest the subject is told to breathe out forcibly and just as he is about to expel the last of his breath to give a slight cough, then to breathe in immediately, but not far beyond the position of rest. In order that the examination of the upper chest may be made satisfactorily it may be necessary, however, to demand a greater expansion of that part of the chest; but, as a rule, the danger lies in the other direction—that the inspiration will be too deep. The object of exhaling before cough is to reduce the bronchial tubes to their smallest size, so that any moisture that may be present in them will be more likely to be caught and moved by the sudden current of air than would be the case in more widely expanded tubes. The cough has for its purpose to give a final push to the diaphragm and to send a puff of air through the tubes; a slight grunt or catching of the breath is all that is necessary after the subject has obtained an idea of the correct method of breathing. It is important that but one cough should be given. Repeated coughs may prevent râles from being heard. This procedure is used for the detection of râles which do not manifest themselves on ordinary breathing and is espe-

cially in place in early and doubtful cases of tuberculosis. It should not be employed in advanced cases, or should be used with great reserve, because it is a too severe exercise for a seriously damaged lung.

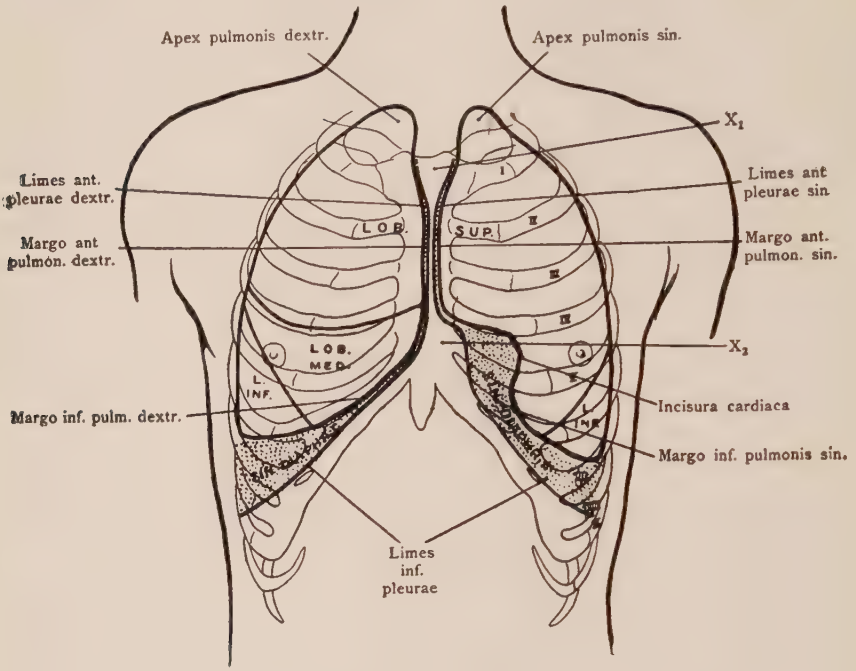


FIG. 18.—Boundaries of lungs and pleura. Anterior view. (Corning, "Lehrbuch der topographischen Anatomie," J. F. Bergmann.)

Breathing of the second type may also be used for the demonstration of some interesting and important phenomena in connection with diaphragmatic breathing.[28] Secure if possible a healthy, young and strong man with good control of his muscles and with sufficient intelligence to comprehend his instructions and teach him the second type of respiration. Let him breathe as in the "expiration and cough" method, ending expiration with a slight cough. Listen with the stethoscope just below the base of the right lung in the axilla. If the subject has been properly trained, sounds will be heard during inspiration which closely resemble medium-sized, moist (sometimes, if respiration is forcible, dry) râles. These sounds, known as "marginal sounds," are heard only over that portion of the lung that expands to fill the costopleural sinus; they are heard but once at a given point during each inspiration. In the beginning of inspiration they are heard in the upper part of the costopleural sinus,

[28] G. E. Bushnell, "Marginal Sounds in the Diagnosis of Pulmonary Tuberculosis," *Med. Rec. N. Y.* Dec. 21, 1912.

farther down near its end. They are heard at the time that the shadow of Litten passes the point of auscultation. Determine the limits of the costopleural sinus from above downwards and, detaching one of the rubber tubes of the stethoscope, slip this tube over one of the arms of the bell of another stethoscope. Close the free arms of the two bells with the thumbs and place the bells, one over the upper part of the costopleural sinus, the other in the next intercostal space below. If the respiration is sufficiently vigorous and not too rapid, it will be perceived that the sounds are advancing from above downwards in a single line and that

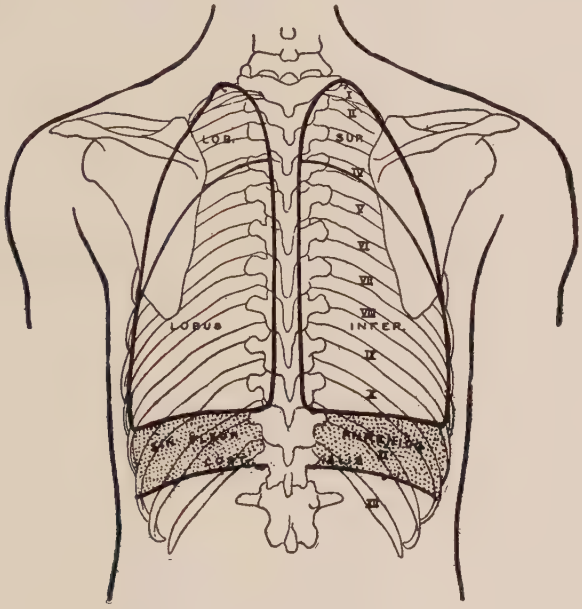


FIG. 19.—Posterior view of the lungs; lower borders of the lungs and pleura. (Corning, "Lehrbuch der topographischen Anatomie," J. F. Bergmann.)

they are heard first by the ear that is connected with the upper stethoscope and only after an appreciable interval by the other ear. When the sounds are well elicited the phenomenon is an interesting one. Numerous rippling sounds are heard—they suggest a wave breaking upon the beach—which are plainly enough moving in a single line. The line comes, passes and disappears; it is heard a moment later by the other ear—still as a single line—the sounds entering the stethoscope, passing through it and disappearing in the same way. If the force of expiration equals that of inspiration these sounds are producible indefinitely. With the untrained subject who leaves expiration to be effected by the elastic recoil of the ribs and abdominal wall they, as a rule, disappear after a few breaths. But if the subject is instructed to exhale a few times more forcibly than he inhales the sounds may be caused to reappear.

Auscultation of the portion of the heart that is not covered by the lung being generally performed during cardiac examinations, the fact is not well known that similar sounds may be heard in this location during inspiration, and especially after expiration and cough, as the tongue of lung known as the lingula pushes its way to the right. The stethoscope should be placed over the region of absolute heart-dullness and the sounds should be observed during an inspiration deeper than usual. The importance of these sounds is solely that they may be confounded with signs of disease. The writer once knew a diagnostician who made point of examining the lingula and almost invariably found evidence of a lesion there in cases of pulmonary tuberculosis! These sounds have the same rationale as the sounds of the costopleural sinus and are to be classed as marginal sounds. The subject of marginal sounds will be taken up again in connection with auscultation of the diseased chest. (See pp. 167–171.)

The Borders of the Lung.—The inner border of the lung is only of importance in connection with the percussion of the heart. On account of the resonance of the sternum as a whole the location of the lung borders is determined with difficulty in this region by percussion. Above in front the lung extends equally on both sides to a distance of 3–4 cm. above the upper border of the clavicle, and in the back to the level of the spinous process of the seventh cervical vertebra. The lower border of the lung forms a line that runs approximately horizontally around the thorax. Beside the sternum it is at the lower border of the sixth rib, in the mamillary line at the ninth and beside the spine at the eleventh rib. Here the writer follows Gerhardt.[29] The location of the lung borders is however given somewhat differently by different authors.

The differences in height between inspiration and expiration is so slight in quiet breathing that it lies within the limits of error in percussion. It is desirable for the student to make it a practice to percuss out the lung borders in their average position, which will be approximately that of the position of rest, and also to determine the position of the lower border of the healthy lung in full inspiration and in forced expiration. The difference in height of these lines at the anterior axillary line in good breathers will be from 3½ to 4½ inches.

The level of fullest inspiration is lowered on the superior side and raised on the inferior in lateral recumbency in a horizontal plane. If the subject breathes as deeply as possible, the superior lung descends to fill the costopleural sinus of the superior side. According to Gerhardt, it is only when the subject lies upon the side that inspiration completely fills the costopleural sinus of the superior side at its deepest part where

[29] *Loc. cit.*

it is bisected by the axillary line. After the upper lung has thus expanded it moves but little in ordinary quiet breathing, the inferior lung performing most of the work of respiration.

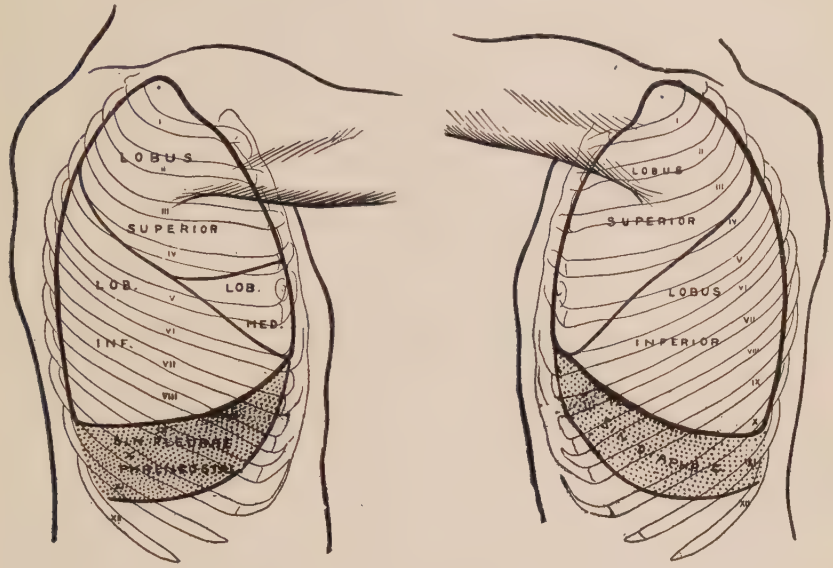


FIG. 20.—Boundaries of right lung and lower pleural borders seen from the right.
FIG. 21.—Boundaries of the left lung and lower pleural borders seen from the left. (Corning, "Lehrbuch der topographischen Anatomie," J. F. Bergmann.)

The behavior of the superior lung in this test gives important information as to the presence of pleural adhesions. This method should, therefore, be studied upon the healthy subject. In such study, mark the level of fullest inspiratory expansion at the base in the axilla while the subject is erect and note the difference on the superior side after a full breath when he lies upon the side. The lung will be found to have descended some two inches below the level of the base of the other side, if the costopleural sinus is free and the lung margins normally mobile. Absence of this descent of the lung usually means pleural adhesions. The right side is more easily examined in this test than the left because of the tympanitic resonance of the stomach on the left side. The extent to which the lung fills the anterior portion of the pleurocostal sinus should also be noted. When the sinus is completely filled the lung should extend nearly to the costal arch. Further details as to the rationale of the production of marginal sounds and the behavior of the diaphragm in lateral recumbency are given in the discussion of atelectasis in Chapter IV.

The Shadow of Litten.—With favorable illumination a shallow hori-

zontal furrow in the complemental space which descends during a deep inspiration may be detected on inspection. This furrow corresponds to the line of separation of the diaphragmatic from the costal pleura and is a purely physiological phenomenon. It indicates the presence of a slight resistance to the separation of the two surfaces, moist with a lubricating fluid which is not without viscidity. The same condition is better shown by the marginal sounds that accompany the separation. They are in fact the auscultatory equivalent of Litten's sign. The Litten shadow is difficult to detect if the layer of subcutaneous fat is thick. If absent in the emaciated, the cause of the absence is usually pleural adhesions which prevent the separation of the diaphragm from the chest wall.

THE DISPLACEMENT OF THE HEART IN DISEASE OF THE LUNGS

The thorax expands and contracts as a whole in normal respiration, the stimulus thereto being of origin in the medulla and the descending nerve fibres decussating in such a way as to insure that the stimulus that initiates the respiratory act shall be communicated to both of its halves. The mediastinum might be defined as the space in the thorax not filled by the lungs. Such a negative definition would serve to emphasize the fact that there is no effective partition wall between the two pleural sacs. And in view of the arrangement of the parts such a partition would be out of place; for the two lungs and the mediastinum are equally subjected to the changes of atmospheric pressure which result from the movements of the thorax. The differences of pressure are equalized by the ingress and egress of air through the main bronchi of the two lungs. The normal balance of this group of organs depends upon the equal expansion of the lungs, the mediastinum being entirely dependent upon the respiratory organs for the maintenance of the equilibrium of the group. It necessarily follows, therefore, that the volume of one lung cannot be increased without a corresponding diminution of the volume of the other lung, except in so far as the increase in volume might be taken care of by a lower position of the diaphragm on the side concerned. Conversely, if one lung is diminished in size the other lung must be correspondingly enlarged, except again in so far as the diaphragm by its rise might offset the diminution.

It is a general law that a change in the volume of the contents of one of the two sides of the thorax results in displacement of the mediastinum toward the smaller side, whether that side is the diseased side or not.

There are two cases under this law: 1. The diseased hemithorax has the smaller content; in this case the deviation is towards the lesion. 2. The diseased hemithorax has the larger content; in this case the deviation is away from the lesion.

1. The diseased hemithorax has the smaller content. There are two causes for the failure of the affected lung to fill the hemithorax: (a) The lung is absolutely reduced in size; it no longer fills the space within the chest that it should occupy: (b) The lung does not differ materially in size from the normal when at rest but, having lost a portion of its elasticity, it is no longer able to expand fully in inspiration. During inspiration, therefore, the lung is smaller than it should be.

The most purely uncomplicated illustration under (a) is afforded by massive collapse of the lung. Massive collapse of the lung is a condition that was observed frequently during the late war. It may occur contralaterally—that is, the sound lung may collapse when the other lung has been injured—or it may follow injuries that do not implicate the thorax at all—non-penetrating wounds of the abdomen, wounds of the pelvis, buttocks and thighs. We may assume then that when the affection develops in a previously healthy soldier the lung that collapses contralaterally was a perfectly healthy lung before the collapse occurred. The physical signs of the entirely collapsed lung are those characteristic of an airless lung. There is necessarily a very considerable diminution in the volume of the lung. The affected side of the chest is retracted and immobile; the ribs are closer together than is normal; the diaphragm occupies an extraordinarily high position. The heart is displaced toward the affected side; if the left side is affected the heart impulse may be felt in the axilla, if the right in the right nipple line and it may be as high as the third rib, showing the unusual height of the diaphragm. The displacement of the heart, Sir John Bradford says,[30] is the most important and characteristic sign of collapse of the lung. Here the sudden loss of volume creates a strong negative pressure; the neighboring organs are crowded into the empty space, both heart and diaphragm being displaced toward the affected lung to the fullest extent that the anatomical conditions permit. The moving force is of course the atmospheric pressure acting through the healthy lung, which is dilated in the process to the extent required to fill the void, and upon the abdomen.

According to Hoover,[31] in congenital malformations of the bronchi, if the subject survives, there is no deformity of the chest wall, but the picture is that of bronchitis with bronchiectasis. The mediastinum is dis-

[30] Sir John Bradford, "Oxford Medicine," II, 127.
[31] *Idem*, II.

placed toward the affected side and the sound lung is enlarged, or, more correctly, the affected side falls behind the normal side in its growth and the space which should have been filled by the imperfectly developed lung is occupied by the heart and by the hypertrophied lung of the healthy side of the body. Even in this congenital condition, therefore, the compensation is bilateral and is effected by the heart and healthy lung, the thorax apparently developing independently of its contents, so long as the underlying deficiency is adequately compensated for and the intrathoracic pressure is not rendered unequal.

(b) Perhaps the best illustration under (b) of uncomplicated inexpansibility of the normal-sized lung as a cause of displacement of the heart is afforded by bronchial stenosis. Jacobson finds, according to Hoover,[32] the most striking sign of stenosis of a bronchus to be the inspiratory movement of the heart toward the affected side, the displacement being more than 2 cm. as determined by the orthodiascope. During expiration the heart moved away from the affected side—the displacement was therefore rhythmical with the respiration. Here the stenosis of the bronchus had probably caused a complete, or partial, atelectasis of the lung below the obstruction. This was presumably compensated for by a localized emphysema, which would develop quickly in acute and severe dyspnea, so that the volume of the lung remained practically the same when at rest—there was no void to be filled. But the affected lung had lost a portion of its elasticity; it was unable to follow the thoracic movements as freely and fully as in health. The chest wall over the affected area rose, therefore, against a stronger resistance; a greater negative pressure developed and the heart and the healthy lung were sucked over in inspiration to equalize the pressure. When the inspiratory pull of the thorax ceased the intrathoracic pressure became equal on the two sides and in expiration the heart and the healthy lung returned by virtue of their elasticity nearly or quite to their normal position. The healthy lung in such a condition may be said to breathe vicariously for the diseased lung to the extent required by the loss of inspiratory expansion of the latter.

The same principles apply to all infiltrated, indurated or collapsed pulmonary tissue which becomes relatively inelastic and, therefore, relatively inexpansible. The two halves of the thorax tend to rise equally in inspiration since they are actuated by a bilateral stimulus. But the thorax on the diseased side can only expand in so far as it is followed by the lung. The lung responds to the limit of its elasticity and no farther to the traction of the chest wall. If the mediastinal pleura were thickened

[32] Hoover, "Diseases of the Bronchi," Oxford Medicine, II.

so as to become unyielding, or if it were effectively bound down by adhesions, the lung would be isolated and the expansibility of the overlying thorax would be measured by that of the lung. But as a rule the pleura retains its elasticity and yields to pressure. The failure of the diseased lung to respond equally with the healthy lung to the traction of the chest wall is then compensated for to a greater or less extent by a lateral expansion of the healthy lung.

Displacement of the heart toward the lesion is seen most frequently in pulmonary tuberculosis. In tuberculosis there is usually both a loss of pulmonary tissue and a loss of expansibility. The former is the chief factor in producing the cardiac displacements of advanced cases. The actual decrease in the size of the affected lung in so far as it persists, is compensated for by a permanent deviation of the heart and a permanent overexpansion of the healthy lung due possibly in part to hypertrophy, but undoubtedly very largely emphysematous in character. There is, therefore, no pendulum movement of the heart. Rhythmical movements which might be expected in the early tuberculous lesion, though they may possibly be found if looked for radioscopically during forcible respiration, are not characteristic of such a lesion. Their absence is probably to be ascribed to the existence of emphysema, even at a comparatively early stage in the development of a chronic pulmonary tuberculosis.

On the mediastinal surface of the lung, there being no mechanism for expansion, there is necessarily no normal lateral suction. The suction towards the diseased lung is, therefore, exerted upon a part of the lung least accustomed to, and probably least fitted for, active expansion. The result of this is inevitably in time overdistention. It is well known that the lung returns but slowly to its original volume after overdistention from such comparatively insignificant and temporary causes as overexertion in mountain climbing and the like.

When collapse of the air-cells after inspiratory expansion is aided by a positive force other than their own elasticity, as when expansion has occurred against the hydrostatic pressure of a pleural exudate, overdistention would naturally be delayed or prevented; otherwise repeated overdistentions must lead to emphysema. The difference as to pendulum movements between bronchial stenosis and early tuberculosis is to be ascribed in part to the greater chronicity of the latter, in part to dyspneic inspiration in the former. It is quite possible that a displaced heart which is motionless during quiet breathing may be made to oscillate under especially strong thoracic traction.

In advanced cases of chronic pulmonary tuberculosis there is usually considerable loss of volume from contraction and from cavity, and cardiac

displacement toward the lesion is marked. In such cases it has been customary to ascribe the displacement of the heart to traction. It has been supposed that the contracting lung draws the heart to itself by pulling upon the adhesions which almost invariably are to be found on the mediastinal wall. But this view does not account for the presence of the heart in a large cavity the outer wall of which, as is usually the case, consists of little more than the visceral pleura glued fast to the parietal pleura and to the ribs. There is no mechanism here that could develop attraction. Nor is it clear how the heart can be displaced by traction when the pulmonary lesion is small and recent and adhesions, if present, are weak. Yet some degree of displacement of the heart is often to be made out radioscopically at so early a stage as to constitute a delicate index of the existence of pulmonary disease.

Pleural adhesions have, however, a rôle in the deviation of the heart. They fix the lung, thus contributing to prevent pendulum movements. In so far as the contracting lung may exercise traction through adhesions, the effect is to help the displacement toward the lesion of the mediastinum, the only movable lateral wall of the pleural cavity.

While lateral deviation of the mediastinum is the most important factor in compensation for excess of negative pressure from loss of pulmonary volume and from the traction of the thorax upon the inelastic lung, compensation is also afforded by the rise of the diaphragm and the sinking in of the soft parts of the thoracic wall. Wherever the bony skeleton does not offer an effective resistance the soft parts sink in; the intercostal spaces and the supraclavicular and infraclavicular fossae are depressed. In chronic superficial lesions of the lung even the overlying rib sinks under the continued pressure.

If the method of equalizing the pressure within the two halves of the thorax by compensatory expansion of the healthy lung were fully adequate, the sinking in of the soft parts from atmospheric pressure would not be expected to occur, as it does to some extent, even in cases in which there has been no material loss of substance. In view of this fact it would seem probable that the compensation is not perfect and that a certain increase of the negative pressure on the side of the diseased lung, as compared with that of the other side, persists indefinitely. This would perhaps be explained by the fact that the traction upon the healthy lung is exerted, not only against its own elasticity, but also against that of the mediastinal organs.

2. The diseased hemithorax has the larger content. The lungs are normally held in contact with the chest wall by atmospheric pressure which counteracts their elasticity. The intrapleural pressure is negative,

being caused by the elasticity of the lung-tissue. This pressure is equal to −7 to −9 cm. of water in the adult during inspiration and to −3 to −5 cm. in expiration. The negative pressure of inspiration increases with the expansion of the lung, so that it is not a fixed quantity. The elasticity of the lung is, of course, always ready to act so that when because of the presence of liquid or of air in the pleural cavity the contact of the pulmonary and costal pleura is broken, retraction of the lung takes place to the degree permitted by the size of the effusion. In proportion to the amount of elasticity that has been expended in this retraction the bilateral equilibrium which formerly existed is impaired; the pull exerted upon the mediastinum by the healthy lung by virtue of its elasticity, is no longer counterbalanced by an equal pull on the part of the other lung. The mediastinum will, therefore, yield somewhat in the direction of the healthy lung which, expending a portion of its elasticity in effecting the deviation, also retracts to a certain extent and becomes proportionately reduced in volume. This shrinkage of the healthy lung yields more space for the other lung and delays to that extent the further retraction of the latter before the growing effusion. The readjustment made necessary by the presence of the effusion is, therefore, to a certain extent bilateral. The early occurrence of heart displacement in cases of pleural effusion thus receives an explanation. The differences of volume of the lungs thus produced are of no importance except as relates to diagnosis, the heart displacement serving as an index of the existence of effusion.

In small effusions the negative pressure which is present is due to the elasticity of the lung which, not approaching exhaustion, exercises a pull upon the fluid contents of the pleural cavity, as it did under normal conditions upon the thoracic walls. As the exudate increases, however, and the elasticity of the lung becomes more and more exhausted by retraction, the diaphragm is no longer drawn up by the lung nor does the latter help to support the weight of the effusion. The result is that the diaphragm becomes depressed to the position of inspiration, a position which it maintains for a period the length of which is governed by its muscular development and by the amount of effusion. Ultimately, if relief is not afforded the diaphragm bellies downward, reversing its convexity. So long as the diaphragm remains flat, its contraction in inspiration tends to narrow the costal arch and hence to diminish the capacity of the lower part of the thorax on the affected side. According to the laws of hydrostatics the liquid within the pleural cavity exercises a lateral pressure upon the walls of its container. This pressure is resisted by the ribs, but the intercostal spaces bulge outward. The effect of this lateral

pressure is most marked on the side of the mediastinum, the only wall of the pleural cavity that is not guarded by bone or by muscle. The heart is, therefore, pushed over to a certain extent by the lateral pressure of the exudate. If the effusion of liquid or of air continues until the elasticity of the lung and the mobility of the contents of the mediastinum have been completely exhausted, the pressure within the pleural cavity would be expected to become positive. But, singularly enough, an actually positive pressure is not necessarily met with even in cases of rapid and great effusion. Gerhardt, for example, according to Nitsch,[33] found that the pressure in the pleural cavity in recent exudative pleurisy was almost always negative and was entirely independent of the size of the effusion and the degree of dyspnea (that is, it did not disappear when the effusion was large or the dyspnea great); for it was precisely in the largest fresh exudates with severe dyspnea that he had abnormally low negative findings. The elastic retractibility of the lung having become entirely, or almost entirely exhausted, some other factor must now be responsible for the maintenance of such low pressure in an actually overfilled cavity. This can be nothing else than the force of respiration.

In inspiration the diseased side rises to the limit of its expansibility, which is the less the greater the effusion and the greater, consequently, the inability of the lung to follow the movements of the thorax. Under the bilateral stimulus from the medulla the affected side of the thorax, during the early days of the disease especially, strives to accompany the movements of the healthy side. It, therefore, pulls strongly though ineffectively upon the lung, creating a marked negative pressure. This phenomenon will naturally be the more marked the stronger the respiratory movements—the negative pressure will be greatest when dyspnea is most severe.

When exudate has rapidly increased in a previously healthy pleural cavity the diaphragm, having become separated from the lung, has no means of maintaining its normal upward convexity and a weight to which it is not accustomed rests upon its upper surface. Its contraction flattens it and on relaxing it remains flat. But the weight of liquid, if great, may be unendurable, especially in the relaxation of expiration, tending, as it does, to cause the muscle to sag downwards and exerting more pressure upon the heart and upon the abdominal organs. The lateral hydrostatic pressure is also burdensome to the heart, whichever side may be involved. The only relief possible is that afforded by the thorax which, in expanding strongly, lifts a portion of the weight from the diaphragm and relieves the sense of oppression. But this relief is only

[33] Nitsch, *Beitr. z. Klin. d. Tuberk., Würzb.*, 1911, XVIII, 11.

operative during inspiration. Hence the patient after exhaling is compelled to inhale again deeply at once and thus a severe dyspnea results which is really due to his efforts to sustain the effusion constantly by the action of the muscles of respiration. The varying tolerance of the patient for effusion, particularly for rapidly forming effusions, explains the differences with respect to the presence, or to the degree, of negative pressure found by different observers.

The effect of the negative pressure is to draw the mediastinum to the diseased side. Greene [34] reported in 1906 the discovery of a rhythmic lateral displacement of the heart as a sign of unilateral pleural exudate, the heart being drawn toward the affected side in inspiration and moving away from it in expiration. The extent of the movement was sometimes as much as two inches. The phenomenon was best observed through the fluoroscope and was only obtainable when the respiration was deep.

Saugmann reports a case of artificial pneumothorax in which he observed through the fluoroscope that the liquid exudate (one of several months' standing) rose and that the mediastinum was drawn toward the affected side in each inspiration. He also states that he had noticed in several patients that this phenomenon did not occur so long as the patient was able to breathe almost entirely with the diaphragm, but appeared at once when the breathing became costal.[35]

The observations of Saugmann afford an illustration of the general principle that loss of expansibility of the lung leads to an increase in negative pressure during inspiration; the diseased half of the thorax exercises a suction through the mediastinum upon the lung of the other side. This principle is operative as truly in the case of pleural effusions as in case of parenchymatous lesions of the lungs, but the increase in the contents of the pleural cavity and the hydrostatic pressure tend even more strongly to displacement of the mediastinum in the direction of the other lung. Thus is usually disguised the fact that respiration here also is exerting, or is capable of exerting, a suction which if conditions were different would move the mediastinum in the opposite direction.

In time the respiratory system of the patient adjusts itself to the new conditions. In chronic cases there is no marked dyspnea, the pleura thickens and becomes unyielding and the respiratory rise of the thorax may be no greater than is required by the conditions. In such cases whether the pressure within the pleural cavity is positive or negative will depend in part upon the behavior of the diaphragm, in part upon the

[34] Greene, *Am. J. M. Sc., Phila.*, 1906, CXXXI, 519.

[35] Saugmann and Hänsen, *Beitr. z. Klin. d. Tuberk, Würzb.*, 1910, XV, 322.

amount of effusion. If the diaphragm sags beneath its load and the effusion is not too large in amount the pressure may remain negative.

In cases of pneumothorax, whether complicated by liquid effusions or not, the same law holds as in other thoracic affections. In small localized pneumothoraces, the pressure of the contained air being taken up by the elastic retraction of the underlying lung, the condition is one rather of diminished expansibility than of increased bulk. The deviation of the heart is, therefore, toward the affected side. In larger pneumothoraces the bulk of the affected side is increased and the heart is pushed toward the healthy side. Under favoring conditions the mediastinum is drawn back toward the side of the pneumothorax during inspiration. The traction ceases at the end of inspiration and the original causes of the displacement again become operative, so that a movement of the mediastinum in the opposite direction occurs. That is, during inspiration the mediastinum moves toward, during expiration away from the lesion. Thus is produced what is called a pendulum movement, the mediastinum swinging back and forth synchronously with the breathing. In some cases of pneumothorax this oscillation is marked, in others it is barely perceptible; in many cases it does not occur at all. According to Barjon, a moderate, not very high, positive pressure in the pneumothorax and entire freedom of the mediastinum from adhesions are essential conditions for the pendulum movements.[36] Since in such an oscillation movement toward the lesion is due to inspiratory suction, the return of the pendulum to the positive pressure of the pneumothorax, manifestly there is needed a sufficient positive pressure to push it away from the lesion during expiration, but not so high a pressure that the negative pressure of inspiration shall be unable to prevail against it in inspiration.

Pendulum movements of the diaphragm, while not closely related to the cardiac displacement of pneumothorax, should be considered in this connection because of the part that the diaphragm plays in the adjustments for changes of volume of the lung.

If inspiration in health is purely thoracic and the diaphragm is relaxed, the abdominal wall sinks in inspiration. This means, of course, that a part of the suction of inspiration has been satisfied by the rise of the abdominal organs which push the diaphragm upward. The drill-sergeant who wishes to teach the recruit to throw out his chest will tell him to "suck up his guts" when he inhales, thus showing an apprehension of the cause of the hollowed abdomen which exaggerates by contrast the protuberance of the chest of his pupil. Such a diaphragmatic movement is a paradoxical movement; that is, an organ which would be expected to

[36] Barjon, "Radio-diagnosis of Pleuro-pulmonary Affections," 1918, 69.

descend in inspiration and to rise in expiration, actually reverses this process.

When the subject lies horizontally upon the side the superior half of the diaphragm, takes little part in the respiration. Here moderately deep breathing has dilated the superior lung at the outset through the suction of the abdominal viscera falling to one side, that is, downward. Inspiration has been, for a moment, more than usually easy; but, on the other hand, the help once afforded in expiration can no longer be given. There is no longer abdominal resistance to be overcome in the descent of the diaphragm and consequently there is no elastic recoil in expiration; the diaphragm is no longer pushed up by displaced viscera which are returning to their position of rest. The diaphragm, therefore, remains flattened and, unless strongly stimulated, inert. (See p. 170.) It is well to keep this normal picture in mind in approaching the study of the diaphragm in disease, for it enables us to realize how passive the rôle of the diaphragm is.

We have spoken of the inspiratory rise of the diaphragm as a paradoxical movement. This term is, however, generally applied to what are known as the balance-movements, also called the paradoxical movements of Kienböck. The paradoxical movements consist in a rise of one half of the diaphragm while the other half descends, like the scales of a balance. In pneumothorax the diaphragm is separated from the lung and, there being no help at hand to enable it to regain its normal convexity after expiration, it remains flattened. If there is in addition a positive pressure within the pneumothorax, the result is a more marked flattening and lowering of the diaphragm. Because of the collapse of the lung the traction of the thorax can not be satisfied by the admission of air through the main bronchus. There is therefore a negative inspiratory pressure in the pneumothorax which, if it is strong enough to overcome the positive gaseous pressure, results in a lifting of the flaccid half of the diaphragm coincidently with the normal descent of the other half. When inspiratory traction ceases the positive pressure again asserts itself and the diaphragm returns in expiration to its former low level, at the same time that the other half rises. The conditions for the paradoxical phenomenon of Kienböck are: complete collapse of the lung, the presence of a not too great positive pressure in the pneumothorax and freedom of the diaphragm from adhesions. These are practically the same conditions as those which are demanded for the pendulum movements of the mediastinum and in fact the mediastinum and the diaphragm often oscillate simultaneously.

Summary.—In unilateral pulmonary disease the hemothorax with the

bulkier content will displace the heart toward the opposite side. This is true whether the larger bulk is due to an unimpaired lung or to a pleural cavity containing air or liquid in addition to a more or less collapsed lung.

Displacement of the heart, in so far as it is due to negative pressure from loss of lung substance, is not accompanied by oscillations in the position of the heart.

Displacement of the heart of an oscillatory character is toward the side with the greater negative pressure in inspiration. Greater inspiratory pressure in one-half of the chest is conditioned by its relative inexpansibility. Inspiratory displacement is, therefore, always toward the lesion. The moving force is the atmospheric pressure exerted through the more expansile lung.

If the diseased lung is not entirely collapsed or not rigidly fixed by adhesions, the displaced heart may be pushed temporarily still farther from its normal position by the traction of the chest wall during forcible inspiration. On the other hand, thoracic traction may cause a movement of the heart against the force that has displaced it, as in pleural effusion.

In short, inspiratory displacement of the heart is always toward the relatively inelastic, and therefore diseased side of the thorax, a non-oscillating displacement toward the thorax-half of the smaller content, whether its lesser size is due to disease or not.

MOVEMENT OF THE COSTAL ARCH AS A MEANS OF DIAGNOSIS

The diaphragm is singularly dependent upon outside assistance for the maintenance of its functions. The return to the convexity of its expiratory position is an entirely passive act, so far as the diaphragm is concerned. Upon relaxation after inspiration it in part follows passively the receding lung, in part is pushed upward by the recoil of the elastic structures in and about the abdominal cavity, against the opposition of which it had made its descent in the preceding inspiration. That the former is the more important factor is shown by the persistence of the convexity of the diaphragm after laparotomy.

In so far as function is concerned, the two halves of the diaphragm each act as a part of the lung of which it constitutes the lower boundary. They are in fact two more or less independent organs in respiration, though anatomically forming a single organ. If in health the diaphragm appears to functionate as a single organ, this is true only on the condition that its two halves follow in expiration two healthy lungs which col-

lapse to an approximately equal degree, and that its two halves are pushed up to about the same extent by the abdominal pressure. The importance of the latter and the degree to which the two halves of the diaphragm may be independent of one another, even in normal respiration, are best shown by the phenomena that manifest themselves in recumbency on one side.

The flaring of the lower ribs and the widening of the costal arch in inspiration are effected by the intercostal muscles. The diaphragm, as a muscle, is much more powerful than the intercostals. But, so long as it retains its convexity, its power to approximate its points of insertion remains in abeyance and the effect of its contraction is simply to diminish its convexity, or to flatten it. In quiet inspiration its activity ceases when its descent is completed. If, however, it continues to be activated after it has become flattened, or if when inspiration begins it is already flat, its contraction overcomes the opposition of the intercostals, the lower ribs move inwards and the costal arch is narrowed. The changes in the mobility of the costal margins and of the lower ribs may of course be unilateral or bilateral, according to the condition present. The direction and degree of movement of the costal margin, in view of the normally nice balance of the opposing muscles, thus becomes a delicate index as to the position of the diaphragm and hence, inferentially, as to the nature of a morbid process which is situated in proximity to the diaphragm, whether above or below it.

A subphrenic abscess, for example, will push up the diaphragm on the affected side, thus increasing its convexity; the costal margin of this side will move outward more freely than that of the other side of the chest. On the other hand, a good-sized empyema will depress the diaphragm; its activation will usually draw the costal margin inwards. Hoover cites a case which shows that the determining factor in the mode of action of the diaphragm is not in its elevation or depression but its shape and that convexity of the diaphragm downward operates in the same way as its more usual upward convexity to place the organ at a mechanical disadvantage in comparison with the intercostals in controlling the respiratory movements of the lower thorax. In the case of a patient with large empyema of the left pleural cavity the costal margin of the left side moved outward in inspiration, though not so far as the right costal margin. Two pints of pus were removed by aspiration, after which the costal margin moved strongly toward the median line in inspiration. Three more pints of pus were then aspirated and again the left costal margin moved away from the median line during inspiration. Here the diaphragm was at first depressed by the weight of the exudate

so as to be convex downward. The first aspiration left it flattened; the second permitted it to regain its normal upward convexity. Only while the muscle was comparatively straight could it gain control of the costal margins and draw them toward the median line in inspiration.[37]

Excursion of the costal borders should be studied only when the patient is lying down with all abdominal muscles relaxed. The examiner applies his thumbs along the costal margins at symmetrical points on the two sides. Care must be taken to differentiate movement upward and forward from movement in relation to the median line. It is only the latter that has to do with the balance between the diaphragm and the intercostals.

[37] Hoover, "Oxford Medicine," II.

CHAPTER IV

PHYSICAL DIAGNOSIS OF DISEASES WHICH IMPLICATE LUNGS AND PLEURA

PNEUMONIA

If a patient has a chill followed by fever, a stabbing pain near the nipple, rusty expectoration and a flushed cheek, there is hardly a necessity for the study of physical signs in order to make the diagnosis of lobar pneumonia. But the disease does not always by any means pursue a typical course and all of the symptoms mentioned are not necessarily present. A frank pneumonia, running its course quickly through hepatization to resolution, indicates a relatively high resistance and, consequently, a good reaction to the attack of the pneumococcus. A slow, imperfect and irregular development of pneumonia and a lingering course, on the other hand, denote, as a rule, a less good reaction and a more doubtful prognosis. It is in this class of cases that physical diagnosis is most difficult and at the same time most necessary for a proper understanding of the course of the disease.

As has recently been shown by experiment, pneumonia probably usually takes its rise from the glands of the hilus and works out from the central to the peripheral portions of the lobes involved. The case may be seen before the inflammatory engorgement has reached the surface of the lung, in which event the only sign is slight dullness from the impairment of the vibratility of the lobe as a whole, elicited by strong percussion; the percussion-note of the affected side will be higher pitched and of less duration than that of the other side. This finding hardly justifies a positive diagnosis. But soon faint and distant râles are to be heard and the breathing becomes slightly bronchovesicular. This change in the breath-sounds may be detected at an earlier time by immediate than by mediate auscultation. When the engorgement has progressed to the surface of the lung the appearance of crepitant râles removes such doubt as may have existed as to the diagnosis. If these râles are not heard on ordinary breathing they may sometimes be brought out by cough followed by a deep inspiration. The breathing is now distinctly bronchovesicular vocal resonance changes to bronchophony, fremitus is increased locally, the percussion-note is dull, or there may be tympanitic

resonance due to the presence of both liquid and air within the air-cells. With complete hepatization the crepitant râle of course disappears. The solidified lung conducts the sound of râles originating in the patent bronchi behind the consolidated area. These râles are medium-sized and coarse consonating râles, more or less abundant according to the degree of the accompanying bronchitis. The breathing will now be purely bronchial, the breath-sounds being made up entirely, or almost entirely, of the relatively high-pitched tones that undergo reinforcement in the bronchi. Well-marked bronchophony is now present, and, it may be, changes into pectoriloquy. Whisper also may be abnormally well conducted. The percussion-note is now very dull. Such is the picture of a completely hepatized area.

The examiner should not content himself with the simple diagnosis of the existence of pneumonia in the stage of hepatization. The extent of the involvement also should be studied as carefully as the strength of the patient will permit. When the entire lobe is not involved bronchovesicular or vesicular breathing may be heard at the borders of the consolidation. If the lesion is spreading, crepitant râles may indicate the newly involved zone. The boundaries of the lesion, as shown by percussion, will be diffuse within the affected lobe, sharp when the interlobar fissure is reached, provided that the adjacent lobes are not also implicated.

It is well to use a dermatographic pencil to mark the lines of the lesion within the lobe, or more usually along the bounding interlobar fissure. Any other points at which peculiarities are noted should likewise be marked. It is the writer's practice in topical diagnosis of this kind to make a record of the finding on the spot in a notebook in which the outlines of the chest, front and back, have been stamped, transferring in this way the record made upon the chest of the patient. It will then be possible to follow with some degree of accuracy the changes which take place from day to day in the extent and intensity of the disease process. It is important to record the outline of the relative heart-dullness and the location of the heart impulse at the first examination. But such procedures, however desirable from a scientific point of view, may require abridgment if the patient's general condition is bad. The examiner must often content himself with such information as can be gathered by a hurried examination of the parts of the chest that may be most easily accessible.

When gray succeeds to red hepatization and the fibrinous exudate begins to be absorbed the subcrepitant râle becomes prominent, the crepitant râle, "crepitatio redux," may again be heard and other râles larger than these two râles are also present; breath-sounds cease to be purely bronchial and again become bronchovesicular and finally normal and the transmis-

sion of voice-sounds grows less until normal limits are reached. Such regressive changes, while sometimes very rapid, do not usually advance with an even step throughout the lesion. Just as in the beginning some parts of the lung are solidified in advance of others, so all parts are not equally ripe for the dissolution of the exudate. And so it comes about that one who goes over the lesion point by point will find differences in the signs at some of the points examined which would be difficult to understand if he had not studied that same region topographically at the beginning of the disease.

If, as is more frequently the case, the pneumonia has involved a lower lobe, the pneumonic lobe, being larger than normal while stiffened with exudate, encroaches upon the corresponding upper lobe, the tension of which is consequently relaxed. As a result of this relaxation the percussion-note of the upper lobe may acquire a tympanitic character which in this instance is not evidence of disease of the part.

While the pneumonic process is still localized and small the loss of expansibility of the affected lobe tends to produce a displacement of the heart toward the lesion. Later in the disease when the hepatized lung has an increased volume it tends to push the heart away from the lesion. But such displacements are usually not well marked.

The percussion-note over the solidified lung varies considerably. Sometimes it is very dull; again, only slightly dull; at times it contains a "clang"; that is, approaches the tympanitic quality. If the upper lobe is involved in the consolidation the air-column of trachea and main bronchi may respond to percussion with a tympanitic note, Williams' tracheal tone, which changes in pitch with the opening or closing of the mouth, becoming deeper when the open mouth is closed.

Sometimes when the conditions would lead us to expect good conduction of bronchial breathing, of consonating râles and of the voice, all breath-sounds and râles may be absent and the voice transmission may be weakened. This is generally due to a plugging of the afferent bronchi with mucus which a cough may displace at any time and enable all these sounds to manifest themselves. But in massive hepatizations, though the bronchi may be open, bronchophony may fail to be heard, not temporarily, but throughout the period of the existence of the solidification. This is supposed to be due to the fact that the lung is so firmly pressed against the ribs on account of its great distention that its vibrations are damped.

The smaller ramifications of the bronchial tree are almost without exception filled with fibrin in pneumonia. Rarely, in so-called massive pneumonias in which the fibrinous exudate is extraordinarily abundant,

the larger bronchi of the affected lobes are also filled with the exudate. This obstruction leads to the absorption of what air remains in incompletely hepatized groups of air-cells and to their atelectasis. The affected portion of the lung has now become perfectly airless the result of which is that dullness becomes very pronounced, increased resistance of the lung-tissue gives it a board-like hardness to the percussed finger and all respiratory sound is absent. This condition is distinguished with difficulty from large pleural effusion. Absence of Grocco's triangle and of cardiac displacement is in favor of pneumonia. But, unless a coughing fit may chance to expel the plugs and recall the breath-sounds, an exploratory puncture, or resort to the x-ray, may be necessary in order to establish the diagnosis when the case is seen for the first time after the consolidation is complete.

The pleurisy that accompanies most cases of pneumonia is, perhaps, usually dry, but may sometimes result in the effusion of a liquid exudate into the pleural cavity. This exudate is, as a rule, small in amount and of no clinical importance. In pneumonias of the lower lobes the presence of a small amount of liquid cannot be determined by physical signs, but in apical pneumonias, since the liquid necessarily accumulates at the bottom of the pleural sac, a zone of dullness may in some cases be detected at the base of the lung separated from the dullness of the pneumonic lesion by the resonant unaffected lower lobe. A large pleural effusion is quite exceptional in pneumonia; according to Fraenkel, it occurs in one per cent of the cases. For the examiner who has followed the case from the beginning the occurrence of this complication should present no difficulties. Dullness on percussion increases or becomes flatness and the transmission of breath-sounds and of the voice is impaired below the level of the effusion. But the impairment in transmission of sound is not so great in pleural effusion complicating pneumonia as in primary pleurisy, for the reason that the solidified lung is a much better conductor of high-pitched sounds than the healthy lung and, as an acoustic medium, differs less from water than does the healthy lung, so that there is less loss of sound by reflection. The changes in physical signs due to the interposition of the effusion will, therefore, be less marked and the amount of liquid present is likely to be underestimated. The distinction between serous effusion and the pus of an empyema is made by exploratory puncture, not by means of the physical signs. The development of an empyema is, however, made probable if coincidentally with the appearance of the effusion there is a marked rise in the body temperature.

The development of lobar pneumonia as a complication of an existing pleurisy with effusion is an extremely rare event. The occurrence of

such a complication would be probable if with the appearance of chill, fever and rusty sputum, the physical signs of a consolidation behind the effusion became apparent; that is, if vocal resonance and breath-sounds, especially bronchial breathing, were detected over the lung where they previously had been absent, the volume of the effusion not being diminished.

In rare instances, usually but not always in patients of advanced years or low vitality, the stage of resolution does not manifest itself. Bronchial, or bronchovesicular breathing, marked dullness and crackling râles persist, though the temperature approaches or reaches normal. If such conditions continue three weeks or more, the probability is great that an indurating or organizing pneumonia is present. In delayed resolution the examiner must continue the topographical study of the diseased area in order to detect the possible development of two other complications, abscess and gangrene. The former is probably present if there is well-marked localized dullness or flatness with absence or enfeeblement of breath and voice-sounds over an area surrounded by lung which has nearly regained its normal physical signs. Gangrene may occur with abscess or as an independent complication.

In connection with the physical exploration of the pneumonic lobe or lobes, the examination of the supposedly healthy portions of the lungs should not be neglected, in order to discover evidences of extension of the pneumonic process, if such there are, at the earliest possible moment.

The differential diagnosis between pleurisy with effusion and lobar pneumonia is usually easy. In pneumonia the physical signs are those of airless tissues which conduct with freedom the various signs characteristic of the disease; bronchial or bronchovesicular breathing, bronchophony, increased tactile fremitus, especially fremitus obtainable with high-pitched voice-sounds and over thoraces which never yield it in health, the crepitant and subcrepitant râles. The picture in general is that of an assemblage of well-conducted and characteristic sounds. In pleurisy, on the other hand, except for the more or less evanescent friction-rub of the early stage the picture is one of negation, of an obstruction that bars the passage of sound. The displacement of the heart away from the lesion in pleurisy, its nearly normal position, or its slight displacement toward the lesion in pneumonia, is an important distinction in the earlier course of the two diseases. And though a hepatized lung may ultimately cause deviation away from the lesion, the displacement is slight in comparison with that due to effusion. The only sign which the two affections have in common is dullness. In pneumonia the note of percussion is not without a certain resonance. In pleurisy the note is

very dull or flat; the percussed finger experiences a sense of increased resistance; the upper level of dullness may have a characteristic curve. Grocco's triangle of dullness on the unaffected side is often to be made out in pleurisy with effusion; it does not often occur in pneumonia. On the other hand, according to Lord,[1] a strip of relative resonance is sometimes present along the spine on the affected side in pneumonia which is absent in pleurisy.

After complete recovery from pneumonia evidence of its former existence is usually furnished by a relatively dull percussion-note over the affected lobe, due to the adhesions of the complicating pleurisy. Such disability as may exist from this cause does not, however, appear to interfere with athletic activity in vigorous men, and the chief importance of the persistence of signs of a former pneumonia is the possibility that they may be ascribed to a more recent affection of the part and thus go to support a faulty diagnosis.

Pericarditis is a formidable complication of pneumonia and, since it usually develops at the height of the disease, it may be difficult to recognize. Pain over the heart, sometimes intense, but sometimes slight or even absent; tenderness on pressure, especially pressure upward in the epigastrium; the sounds of pericardial friction, the rub sometimes to be detected by palpation, are the physical signs of pericarditis. The possibility of this complication is an additional reason for careful notation of the position of the heart. If there is diminution in the force or disappearance of the heartbeat, if the limits of percussion-dullness are enlarged upward and also downward and to the left, so as to go beyond the heartbeat as previously palpated, and if pericardial friction previously heard disappears and the heart-sounds become less plainly audible, pericardial effusion is in all probability present.

PLEURISY

The onset of pleurisy is usually sudden, with pain, fever and a friction-rub as the earliest signs and symptoms. The pain differs much in severity in the individual cases; sometimes it is absent. It is most frequently felt in the axilla or near the nipple, but may be referred to the shoulder, neck, or even the abdomen. In general, pain is characteristic of the pleuritis of the freely moving lower parts of the lung; it is not so frequent, but is by no means unknown, in pleuritis of the upper lobe. Fever, which is only temporary in uncomplicated cases, is rarely high. The pleuritic

[1] F. T. Lord, *Boston M. and S. J.*, Feb. 12, 1914.

friction-sound is the most characteristic sign of early pleurisy. This friction-murmur, which is usually a scratching or grating sound, is produced by the rubbing together of inflamed pleural membranes which are coated to a greater or less extent with fibrin. However, the character of the sound and its constancy vary greatly. It is usually rather evanescent, disappearing when the liquid exudate has separated the leaves of the pleura at the place of production of the sound. The fact that it is heard does not necessarily exclude the presence of an effusion elsewhere in the pleural cavity. The vibrations of the friction-rub can often be felt on palpation. In rare cases the friction-sound persists for several weeks and may even be loud enough to be audible to the bystanders. Friction-sounds are distinguished from râles by the fact that they are neither brought out nor caused to disappear by cough. Râles are more usually accompanied by bronchovesicular breathing; friction-sounds by normal breathing. Lord describes a pseudopleuritic friction-sound which may be heard during the examination of the back when the patient's arms are crossed, the hands resting upon the opposite shoulders. He ascribes this sound which is heard in normal individuals, to crepitus in the shoulder joint.[2] Friction-sounds may return after the subsidence of the effusion. In examining for friction-sounds care should be taken to exclude marginal sounds. It is also to be remembered in auscultation of the heart that marginal sounds are normally heard when the lingula pushes across the heart in inspiration; they may be mistaken for sounds due to pleurisy or pericarditis. (See p. 122.)

Pleurisy may heal without the development of effusion; in many cases an exudate is present in such small amount that it fails of recognition; in others its increase is extremely rapid. The contact of two surfaces of inflamed pleura leads first to their mutual agglutination by fibrinous exudate and, secondly, to their more or less permanent and extensive union by the organization of this exudate. Localized and thin adherent membranes stretch into ribbons and finally cease to restrain the lung movements. Such adhesions give no physical signs. More extensive and thicker membranes may permanently cripple the lung. Restricted expansion, slight dullness on percussion, enfeebled and often slightly bronchovesicular respiration, slightly increased vocal fremitus and resonance may remain as permanent signs of a past pleurisy or pneumonia, though the health of the individual may be perfect. In such cases the x-ray may give no indication of the presence and location of the pleural adhesions.

Opacity due to past or present pleuritis manifests itself in the roentgenograph to a uniform opacity of the pulmonary field. The opacity is

[2] Frederick T. Lord, "Diseases of the Bronchi, Lungs and Pleura," Phila. 1915, 463.

due either to the thickening of the pleura or to increased congestion of the pulmonary tissues the respiratory movements of which are inhibited by the adhesions. If due to thickened pleura the opacity is permanent, but when the cause is defective lung movement the opacity clears up or notably diminishes after deep respirations. Pleurisy over the apical lung often results in dense adhesions which cover the entire apex, or its upper portion, constituting the so-called "pleural cap." It is in this location that uniform opacities are most frequently observed. But an extensive sheet of thick fibrinous or organized exudate, as well as those tough and firm local adhesions which restrain the lung and sometimes conduct the voice and breath-sounds through an effusion, indicate, as a rule, the past or present existence of parenchymatous lesions, the signs being of course the more apparent the more marked the induration or infiltration of the lung parenchyma. The distinctive feature in the roentgenograph of opacity due to the pleura is its uniformity; parenchymatous lesions are characterized by local variations in density, streaks, spots and cloudy makings; a uniform and dense opacity denotes liquid (serous exudate, blood or pus), or tumor.

So long as the pleural exudate is small in amount it produces no effect upon the physical signs that can be positively recognized. As it grows, dullness is made out with increasing ease. The effusion collects first over the diaphragm, and the pleurocostal sinus may disappear. A zone of dullness may sometimes be recognized at the phrenicocostal angle. The breath-sounds are simply weakened at first, probably on account of the restraint by the patient of respiratory movements in his desire to avoid pain. With an appreciable amount of exudate the breath-sounds above the level of the effusion become slightly bronchovesicular and may be less loud than those at the same level on the other side. A careful comparison with the sounds on the other side of the chest is necessary in order to detect the beginning of this change. Coincidently with the alteration in the character of the breath-sounds there is noted a diminution in the transmission of the voice and in the vocal fremitus. Moist râles are sometimes to be heard through the effusion from moisture in the air-tubes of the lung, due to disturbance of the normal conditions of the lung-tissues.

The postition of the heartbeat should be noted at each visit, displacement of the heart away from the lesion being an important indication of the existence of an effusion. The heart is displaced at an early period in the growth of the exudation and thus indicates the presence of the latter, but the minor degrees of displacement are only to be discovered by the aid of the x-ray. The heart is displaced to a greater extent to the

right in left-sided effusion than it is to the left when the disease is on the right side; authors differ, however, on this point. The subject of displacement of the mediastinum is considered in another chapter. (See p. 129.)

Inspection of a patient with considerable pleural effusion shows that the affected side is relatively motionless in respiration, that in the intercostal spaces below the level of the fluid the normal concavity of the soft parts is diminished or effaced and that the ribs move hardly at all. The cardiac impulse may be seen or can be palpated in an abnormal location. Percussion in such a case will give very marked dullness or flatness with a sense of increased resistance which indicates, as a rule, the presence of liquid. It is difficult to determine the exact level of the exudate, for its

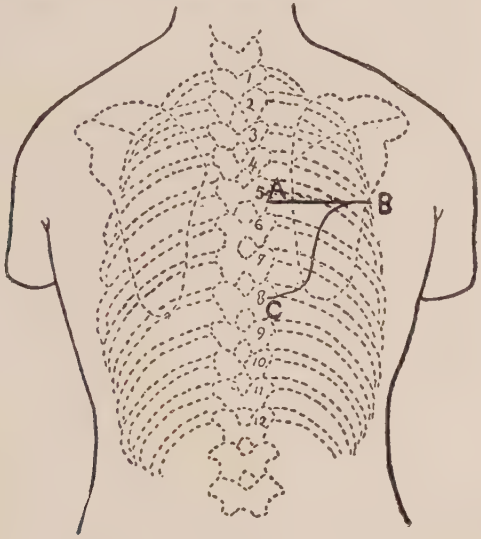


FIG. 22.—The irregular triangular space bounded above by the line A B, on the side by the curve C B and behind by the vertebral column A C is known as the "Dull Triangle" of Garland. (Garland, "Pneumono-dynamics," Hurd and Houghton.)

presence has led to retraction of the lung which normally lay beneath its surface and, as a secondary result, to a relaxation of the lung above the effusion. The relaxed lung above the level of the exudate has a tympanitic percussion-note and the percussory problem here is the difficult one to define the boundaries of an airless mass against tympanitic resonance. Since, as we have already seen, tympanitic resonance invades to some extent neighboring airless bodies, the probability is that the level of the effusion will be placed too low. Except in very large effusions the upper border of the effusion is not a horizontal, but a curved line.

Damoiseau is credited with being the first to discover that the line of

an effusion may be a curved line. Ellis of Boston described the same phenomenon independently. The curve of the effusion is called the Damoiseau-Ellis curve. Garland, also of Boston, has described what is known as "Garland's triangle." Garland furnishes a diagram of the posterior surface of the thorax upon which is drawn the curve of an effusion. He says: "In the diagram it will be seen that I draw a horizontal line, AB, from the summit of the curve to the vertebral column and have thereby enclosed a roughly triangular space, ABC. This space corresponds to the lowest portion of the lung and is especially liable to be obscured by dullness. The lung lies more in contact with the chest wall, but its resonance may be so dull as to escape detection unless careful percussion is made and the patient breathes deeply. I have termed this space the *dull triangle* and its recognition is of vital importance." [3] He states that Heitler of Vienna has observed the same triangular space and that Rosenbach has noticed that the resonance of this portion of the back will often clear upon exercise or by breathing, and makes such clearing up of the resonance of a dull back distinctive between pleurisy and pneumonia. Elsewhere Garland emphasizes the need of percussing lightly, "else flatness will be concealed by resonance transmitted by the lung" and warns against comparing the percussion-note of the two sides while pursuing this inquiry. "The point sought is not the distinction between dullness and full resonance but between dullness and flatness." [4]

The expanded lung will always retract by virtue of its elasticity until that elasticity is entirely exhausted. And, as Garland pointed out, the various portions of the lung differ in their retractile force. This is to be expected for retractility is simply the converse of expansibility. Of course, in the normal expansion of the lung to fill the thoracic cavity, the different portions of its surface are exposed to the same degree of negative tension during rest. But there is a great difference as respects the degree to which the different portions are stretched in the suction of inspiration; there are great changes of volume, for example, in the axillary portions of the base of the lung, very slight changes in the paravertebral portions of the upper lobe. The traction of inspiration is in a direction away from the hilus in all parts of the lung. The direction in which an elastic body is stretched governs the direction in which it will retract when released from traction—the retracting lung retracts toward the hilus. So long as the lung has not expended all its elasticity it will exercise a pull upon whatever substance it may be in contact with in the pleural

[3] Garland, Supplement to Ziemssen's "Cyclopedia of the Practice of Medicine, Diseases of the Pleura," New York, 1881, 273.

[4] Garland, "Pneumono-dynamics," Boston, 1878.

cavity, whether that be diaphragm or liquid or a localized bubble of air. By virtue of its elasticity the lung supports the pleural effusion; the curvature of the effusion is also the curvature of the surface of the retracting pulmonary tissues. The course of this line will depend upon the relative degree of retractility of the various parts of the lung. It may be expected that parts of the lung which are very elastic will retract farther than those which have but slight elasticity. The part of the lung which is exposed to the greatest respiratory traction is the axillary portion of the base or lower part of the lower lobe, for this region is pulled upon both by the diaphragm and by the ribs. It would be expected, therefore, that this region would retract to the greatest extent. On the other hand, the paravertebral portion of the lower lobe is exposed to less traction and would, therefore, tend to retract less. We may say then that the greatest and earliest retraction of the lung would be at the external base and that the general direction of the surface of retraction would roughly be at right angles to a line drawn from this part of the lung to the hilus. Observation shows that this statement is approximately correct.

Observers are now pretty well agreed that the curve of the effusion is highest in the axilla. The fact that Garland's triangle is a "dull" triangle explains the difference of opinion as to the course of the curve of the effusion, as it approaches the spinal column. Some maintain that the curve rises again at this point. But, according to Edens, the x-ray shows that the level of the effusion is correctly indicated by percussion over Garland's triangle.[5] It may be said therefore that the most retractile portions of the lung, those of the axillary region, go farthest in their retraction and that, as the curve of the effusion approaches the median line in front and behind, and consequently reaches less extensible portions of the lung, it descends. One point is to be kept clearly in view which is that so long as the Ellis curve is to be made out the elasticity of the lung is not exhausted, but governs the shape of the surface of the underlying fluid and that, so long as this is the case, diminution of the volume of the lung has not been due to compression, but purely to retraction.

The curve, as described, is only found in the non-adherent and previously healthy lung. Since the curve is due purely to the retractility of the lung, anything that influences unfavorably that retractility, or, what amounts to the same thing, the elasticity of the lung, such as pleural adhesions, parenchymatous foci, fibrosis etc., will alter the form of the curve. Any change of posture which changes the shape of the lungs must affect the curve. Alternations between the vertical and the hori-

[5] Ernst Edens, "Lehrbuch der Perkussion und Auskultation," Berlin, 1920, 194.

zontal positions are generally said to have little effect upon the level of the effusion in the closed pleural cavity. This assumes that the elasticity of the lung is still operative. However, Garland states that the curve in its purity is only found in the vertical position. This position should, consequently, always be used in studying the curve. With very large effusions the elasticity of the lung is, of course, too slight to maintain the curve. When air is admitted into the pleural cavity the lung retracts completely, exhausting its elasticity, and is no longer able to control the effusion which then is horizontal in all positions of the body.

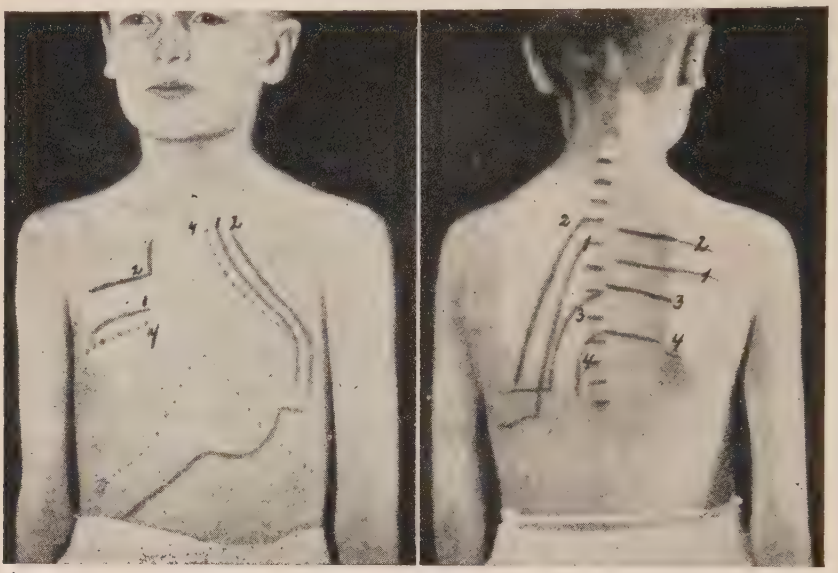


FIG. 23.—Grocco's or Rauchfuss' triangle. 1–4, borders of dullness when first seen and during the course of observation. (Edens, "Lehrbuch der Perkussion und Auskultation," J. Springer.)

Grocco's triangle of dullness on the unaffected side of the chest as a sign of pleural effusion was first noted by Koranyi but was discovered independently and more fully described by Grocco. According to Fishberg,[6] Grocco thus described it in his first communication on the subject: "Paravertebral triangle of the side opposite to a pleural effusion. When, with a pleural effusion of sufficient size one percusses from above downwards along the spinous processes of the vertebrae with the patient in a sitting posture there appears at the level of the fluid a dullness which, relative at first, becomes absolute as one passes downward, associated with a progressively increasing sense of resistance. In like manner, in percuss-

[6] M. Fishberg, "Tuberculosis," Phila., 1922, 493.

ing downward on the healthy side along lines parallel to the spinous processes, there is noted, opposite the dullness in the median line, a paravertebral area of deficient resonance of triangular shape. One side of this dull area is represented by the line of the spinous processes; another by the lower border of the area of thoracic resonance for a short distance, which varies in length from two to three or more centimeters; the outer side is represented by a line which, starting from the base, rises obliquely to unite at an acute angle with the median line at about the upper limit of dullness. In a right-sided effusion, other things being equal, the paravertebral triangle has seemed to me more marked."

The view that the presence of Grocco's triangle is a proof of pleural effusion, as contrasted with pneumonia, is not to be accepted absolutely, since several observers have noted it in pneumonia.

Pleural effusions on the left side result early in the disappearance of the tympanitic resonance of Traube's semilunar space. (See p. 112.) The absorption of the exudate may first be recognizable by the reappearance of the tympanitic note. As has been remarked, it is difficult to determine the lung boundary when the percussion-note is tympanitic. This may, however, be done by percussion behind Traube's space where the lung borders upon the spleen. In right-sided effusions downward displacement of the liver is one of the signs of pleural effusion.

If the patient is a man with a strong and deep voice, tactile fremitus will indicate the level of the effusion with considerable accuracy, the fremitus being absent or weak below that level and suddenly increasing in strength beyond the normal above it. But, although the timbre and pitch of the voice may be favorable, this test fails to give well-marked and satisfactory findings if there chance to be a thick layer of adipose tissue, dropsical thickenings of the soft parts, or chronic inflammation of the pleura. The breath-sounds are absent or weak below the level of the exudate, partly because the thorax moves but little in respiration, but chiefly because the lung no longer lies directly beneath the stethoscope. Moreover the effusion introduces a new acoustic medium, so that sound is lost by reflection from its surface, as well as by diffusion through its mass. With large effusions the breath-sounds, if heard at all, will be purely bronchial or bronchovesicular with a very marked bronchial quality, their character depending of course upon the amount of air remaining within the lung. It is said that the normal vesicular murmur is almost never heard through pleural effusions. With very large effusions the bronchi are also compressed and the lung is silent. Voice-sounds are usually much diminished or absent over the effusion. If they are well transmitted over any localized area the presumption is that adhesions are

present and the same considerations apply to the occasional local persistence of vocal fremitus and resonance. Whisper is usually absent over large effusions. When heard it probably indicates either transmission through adhesions or parenchymatous lung lesions. Bacelli's sign, the transmission of whisper through serofibrinous but not through purulent effusions, is not trustworthy. Above the level of the effusion the breath-sounds are characteristic of the relaxed lung. On account of the diminished air-content of the parenchyma high-pitched sounds will be conducted more perfectly and the breath-sounds will have a bronchovesicular quality. For the same reason the vocal resonance and fremitus are increased. The bronchophony often takes the character of egophony. Whisper is the more likely to be transmitted the more nearly the tissues approach complete airlessness. Small to medium-sized râles are not uncommon in the lung immediately above the level of the effusion. They are probably to be ascribed to the presence of liquid in the bronchioles from disturbances of the circulation. In cases in which a pleurisy has supervened upon an existing tuberculosis of the lungs, it is sometimes difficult to decide whether the râles that may be heard are to be explained as above or are due to the tuberculosis. If a distinction is attempted it may be said that medium-sized râles, sparsely disseminated, speak for pleuritic, denser clusters of persistent smaller râles for a tuberculous origin.

Inflammation of the diaphragmatic pleura occurs mostly as an extension of the inflammation of the costal pleura in pneumonia of the lower lobes, in tuberculosis, and in extensive or generalized pleurisies. Since the complication often gives no evidence of its presence to percussion and auscultation, it is frequently overlooked. The diagnosis may depend upon the location of referred pain and upon the x-ray. Pain is referred to the abdomen and to the neck. Hyperesthesia and hyperalgesia follow the course of the nerves. The musculocutaneous reflexes are exaggerated over the sensitive areas. Deep pressure increases the pain of abdominal inflammation but is well borne in diaphragmatic pleurisy.

Empyema is more likely to follow infections due to the pneumococcus and the streptococcus than tuberculosis, but is far from unknown in the latter. Collections of pus are more likely to be encysted than are serofibrinous effusions. The region between the diaphragm and the base of the lung is one of the favorite locations of encysted empyemata. In diaphragmatic empyema in addition to tenderness, referred pain, localized basal dullness and the information supplied by the roentgenograph, the movements of the costal arch may throw light upon the collection of pus.

The differential diagnosis between pleural effusion and subdiaphragmatic abscess is sometimes required. The question would hardly be

raised in the case of pleural effusions of relatively rapid formation of which the course has been observed and the history is known. But in chronic and more or less neglected cases doubt may exist as to the nature of a condition which presents an area of dullness in the vicinity of the diaphragm, and in which absence or diminution of breath-sounds and of vocal resonance and fremitus shows a pressure on the lower lung. The discrimination is most easily made by the aid of the fluoroscope. But when this is not available, or as a means of preliminary diagnosis, information as to the condition of the diaphragm may often be obtained by observing the movement of the costal arch. In subphrenic abscess the costal margin of the affected side will move outward freely during inspiration; in empyema its outward movement will be less than that of the other side, or the movement may even be toward the median line.[7] The differential diagnosis between pleural effusion and pneumothorax with effusion will be considered under pneumothorax.

PNEUMOTHORAX

By the term pneumothorax is meant an affection produced by the entrance of air into the pleural cavity and the subsequent entire or partial collapse of the lung. In natural, as opposed to artificial, pneumothorax, air is generally introduced into the pleural cavity through a pulmonary fistula. The cause of the formation of the fistula is, in the large majority of cases, the breaking down of a pulmonary tubercle. Tubercles especially affect the superficial lung and extend to the pleura. If the tubercle caseates and softens a channel may be left which connects the surface of the pleura with a bronchiole or bronchus and thus admits air to the pleural cavity. The fate of the patient after such a fistula has developed depends upon the condition of the neighboring pleura. If the tubercle that caused the fistula was relatively small, if the progress of the tuberculous lesion toward the surface of the lung had been slow and the reaction of the patient had been good, so that pleural adhesions had formed around the tubercle before its rupture, the pneumothorax which results will be small and will be recognized with difficulty. In fact, the very large majority of such pneumothoraces escape recognition altogether. Pneumothorax of this type, being strictly localized, has a very good prognosis. But if the course of the disease has been rapid and the reactive powers of the patient have been weak, effective adhesions may not form and the rupture of the tubercle leads to effusion of air into the general

[7] See: Movement of the Costal Arch as a Means of Diagnosis, p. 134.

pleural cavity, producing a generalized pneumothorax with total collapse of the lung. A third possibility is that the fistula may chance to make its appearance in a region surrounded by a ring of adequate adhesions which owe their origin to some earlier inflammation. A pneumothorax is then formed, larger than that of the first class, but not generalized, as in the second class—encysted pneumothorax. Often the air escapes from the fistula into the general cavity of the pleura, yet already existing adhesions prevent considerable portions of the lung from collapsing. The result of this complication is practically the same as in the case of the regularly encysted pneumothorax—the lung does not collapse as a whole, the pneumothorax is a limited one, and the prognosis is good in proportion to the extent of the pleural adhesions. It is fortunate that, as a rule, the tuberculous process is attended by a pleural inflammation which shuts off the pleural cavity to a considerable extent, for a generalized pneumothorax is one of the most distressing and fatal complications of pulmonary tuberculosis; but it is a relatively rare complication. In a typical case of this kind the lung is completely collapsed and the mediastinum is displaced toward the sound side. The onset is sudden, often declared by a sharp pain in the affected side; the pain is sometimes very severe. A distressing dyspnea quickly develops, the patient suffering from an intense feeling of suffocation; the breathing is rapid, the pulse rapid and weak; the skin, bathed in sweat, is pale or cyanotic. The attack generally terminates fatally within a few days. In less severe cases pain is slight or absent and dyspnea is not marked. In some cases the condition is latent so far as symptoms are concerned and may first be discovered during physical examination.

Pneumothorax sometimes develops suddenly in apparently good health. It is then called spontaneous or idiopathic pneumothorax. The cause of the perforation of the lung is frequently obscure, but it is probably due to the tearing of an old adhesion from straining or violent coughing. The breaking down of a small subpleural tubercle may occasionally be the cause. If the pleural cavity is free from adhesions a generalized and fatal pneumothorax may result. But sometimes these cases run a benign course. It is then probable that the adhesion which has ruptured is not the only adhesion present and that the pneumothorax is more or less localized.

The pulmonary fistula which creates the pneumothorax is usually of small calibre, the result of which is that it very often becomes closed in the retraction of expiration. But it opens again in the succeeding inspiration, valvular pneumothorax. Air is then admitted with each inspiration which can not escape and the air-pressure rises to a considerable

extent. But a needle inserted into the cavity of the pneumothorax to provide a vent for the compressed air does not always appear to relieve the dyspnea. There is undoubtedly a difference in individuals in their tolerance of the same degree of tension which is dependent upon the sensitiveness of their nervous systems to perturbations and also upon the resistance which the mediastinum offers to stretching. We can not, therefore, always explain the individual reactions by the mechanical conditions of fistula and pleural adhesions.

If the fistula does not close in expiration the pneumothorax is called open pneumothorax. In this case there is no rise in the air tension and the patient is relatively comfortable. However, an open fistula is a fistula of large calibre, created probably by the breaking down of a relatively large lesion, so that a pyopneumothorax is the almost inevitable result.

The percussion-note in pneumothorax depends upon the size of the pleural cavity and the tension of its contents. If the tension is not high the note may be tympanitic. A tympanitic quality is often found in the percussion-note in case of open pneumothorax, but its presence is due to the low tension that results from communication with the outer air, not generally to the resonance of connecting bronchi. In some cases the note is tympanitic though the fistula is closed. With higher tension the percussion-note loses its tympanitic quality but remains resonant and loud. If the pneumothorax is large the fundamental tone of the percussion-note may be lower pitched than that of the same hemithorax in health. With high tension the note becomes dull. Incomplete pneumothoraces naturally furnish a great variety of percussion findings according to the extent and the location of the adhesions. It is usually possible to percuss out the adherent areas, these furnishing a duller note than that obtained over the air of the cavity. The percussion-note may have an amphoric quality. The walls of the pneumothorax being invariably smooth, hard and elastic, if the tension is not too high the conditions are favorable not only for the production of the fundamental tone, but also of the high overtones of that tone. Hence metallic sounds, if especially sought for, may sometimes be obtained by percussion.

More or less pleural exudate usually complicates pneumothorax. This, if small in amount, may not be detected by percussion but is to be made out by the splash of succussion. Larger effusions are accompanied by dullness or flatness on percussion, but the level of the fluid is generally placed too low by this method. Characteristic of cavities containing both air and liquid is the horizontal level of the effusion, which persists whatever the position of the patient. This constitutes a means of distinction

of pneumothorax with large effusion from simple pleurisy with effusion. Another point is that in pleural effusion voice and breath-sounds are absent or weak over the effusion and become much louder above its level, while in pneumothorax, except as they may be conducted by adhesions, they are absent or weak both above and over the effusion.

As a rule, breath-sounds are absent or diminished over the pneumothorax. Adhesions may, however, conduct normal or abnormal breath-sounds from the lung beneath. The voice-sounds and the whisper may be conducted in the same manner, but otherwise are absent or much diminished. An amphoric quality may be present in the breath-sounds under the conditions discussed under percussion. (See p. 14.) Metallic breathing, though possible, is rare.

Metallic tinkle is a variety of metallic sound which has usually been ascribed to the sounds produced by drops of liquid falling upon the surface of an effusion. Sometimes the sound appears to be more of the nature of metallic râles. It is probably produced by the bubbling of air through the effusion. In any case it is always to be remembered that the metallic sounds are the overtones of the fundamental tone of the cavity, so that if the metallic tinkle is heard the conditions are favorable for the production of the other metallic phenomena. The coin-sound has already been discussed. (See p. 104.) This sound is found almost exclusively in pneumothorax, but is not peculiar to that condition. As in the case of the other metallic sounds, all that is required for the production of these high-pitched overtones is a cavity of some kind with vibratile walls and containing a volume of air under proper tension and large enough to have a fundamental tone of the proper pitch. The conditions are best satisfied, as a rule, in pneumothorax, but the coin-sound and the other metallic sounds are occasionally heard over large pulmonary cavities.

The diagnosis between pneumothorax and large pulmonary cavities is, as a rule, to be made from the history of the onset. Sudden development of pain and dyspnea is characteristic of pneumothorax. Large cavities have a long history. The typical signs of pneumothorax, the absence of breath-sounds, a positive coin-test, the metallic phenomena, the horizontal level of the liquid, create a strong presumption at least in favor of pneumothorax. There may be doubt as to the diagnosis if a spontaneous pneumothorax is discovered for the first time in the course of an examination. Pneumothorax due to a minute tubercle or to the rupture of an adhesion takes its origin almost always in the upper lobe, for it is in this location that the tubercles and adhesions of obscure incipient or obsolete tuberculosis are likely to be situated. In such cases the base of

the lung will, as a rule, be clear and the lower lobe will contract completely when air collects in the pleural cavity. A clear base, therefore, speaks for pneumothorax. For however large the pulmonary cavity, it is rare that the entire lower lobe to the very base is implicated; there is almost always a remnant of lung at the base in which breath-sounds, bronchial or bronchovesicular, may be heard and which gives a zone of more or less impaired pulmonary resonance on percussion. Such a condition speaks strongly for the diagnosis of cavity, though not of course absolute proof of its existence. Absence or diminution of breath-sounds and of voice transmission in pneumothorax makes a strong contrast with the usual signs of cavity, but an open fistula may conduct breath and voice-sounds to the pneumothorax.

An open fistula means certain infection of the pleural cavity. The fluid that collects in this case is frankly pus. The pulmonary fistula is not large enough to drain this collection thoroughly. On the other hand, the purulent contents of a cavity are often mingled with considerable mucus and sometimes the mixture is of considerable viscidity. If expectoration is of this nature and is raised with comparative ease and in abundance, and if in certain positions of the body it may be drained away completely, there can be hardly a doubt but that it originates in a pulmonary cavity. Pneumothorax sometimes develops in advanced cases of pulmonary tuberculosis in which a cavity, or cavities, are known to be present. In such cases the pneumothorax usually forms over the lower lobes while the cavities are situated higher up in the lung. Here there is generally a sudden appearance of a dyspnea which may be annoying but is rarely severe. The percussion-note of the pneumothorax has a pitch which of course varies with the size of the collection of air and its tension. But the quality of the note will be different from that over the cavity. It is perhaps generally dull-tympanitic in the early history of the pneumothorax.

It is well to make the diagnosis of pneumothorax by physical exploration and the examiner is often compelled to depend upon this means of diagnosis. There is no disease, however, in which the x-ray is of more assistance in the diagnosis and whenever possible the conclusions reached by means of physical signs should be checked up by the fluoroscope and the roentgenograph, the former being essential for studying the pendulum movements of the mediastinum and of the diaphragm. The roentgenograph of a good-sized pneumothorax shows an unusually clear field without lung markings. The lung is seen retracted toward the hilus, or irregularly held in contact with the ribs by adhesions. Liquid if present has a horizontal level, remaining horizontal in alterations of

the posture of the patient, but the liquid within a cavity has also a horizontal surface. The examination for liquid should be made with the patient in the erect posture. The diaphragm is more likely to be flattened in pneumothorax than in cavity. The heart and trachea are displaced toward the opposite side of the body in pneumothorax, toward the lesion in cavity. The presence of lung-tissue below the collection of air speaks for cavity, a clear base for pneumothorax. An encysted pneumothorax, if located above the base of the lung is distinguished from cavity with difficulty by means of the x-ray. With anteroposterior illumination a small pneumothorax does not appear as a clear field unless it is situated on a lateral surface so that its base is approximately perpendicular to the eye of the examiner. Turning the patient so that the illumination is oblique will sometimes furnish valuable information in the fluoroscopic examination.

Small Pneumothoraces.—Barlow and Thompson (Hyg. Bull. No. 132, U. S. P. H. S) have collaborated in a prolonged study of small pneumothoraces, Barlow making the physical examinations and Thompson working in the x-ray laboratory. Their researches in this very difficult subject are so recent that the medical profession has not as yet had the opportunity to confirm them. The utility of the method in practical diagnosis may, therefore, be said to be still *sub judice* at the date of this writing. The following brief account of the physical signs which characterize small pneumothoraces and of the x-ray aspects of the subject is abstracted from their work.

A small pneumothorax makes neither additions to, nor subtractions from the tissues of the part concerned. The physical signs are modified in four ways by the presence of a small pneumothorax: first, by the formation of a more or less flat and shallow smooth-walled cavity immediately beneath the pleura; second, by the more or less complete collapse of the lung-tissues underlying the pneumothorax; third, by the deviation of mediastinal organs to the side of the pneumothorax; fourth, by secondary changes brought about by its presence, such as more rapid healing and fibrosis, reflex disturbances, etc.

On inspection the trachea is sometimes seen to be deviated toward the pneumothorax, or the displacement may be detected by palpation. If there are pneumothoraces on both sides the trachea may be deviated toward the side most affected by the pneumothorax; that is, to the side on which the expansibility of the lung is most diminished. The coin-sound is rare and of brief duration in small pneumothoraces, disappearing when exudate collects. When present it indicates either a moderately large,

or very recent pneumothorax. Tympanitic resonance is found in superficial pneumothoraces, as a rule, but some show increased dullness almost from the outset. The tympany is high pitched, the cavity as a rule being small. If the pneumothorax communicates with a pulmonary cavity there may be two-pitched tympany. Sometimes there is a dull tympany with exaggeration of the high overtones. Sometimes instead of tympanitic resonance there is exaggerated resonance over the entire region affected by the pneumothorax. Dullness finally results from the deposition of plastic exudate or the collection of fluid. The presence of the pneumothorax permits a corresponding retraction of the underlying lung, which retraction may extend beyond the circumference of the pneumothorax. Such collapsed tissue adjacent to the pneumothorax is generally dull. Interlobar pneumothorax may produce either exaggerated resonance midway between apex and base, or if it is more central, may allow the lung-tissue to collapse against the lateral wall, causing a zone of dullness with resonance above and below; the "suspended dullness" of Dieulafoy.

If fairly free of exudate the smooth walls of the pneumothorax amplify the higher-pitched tones and suppress to a considerable extent low-pitched sounds. The metallic phenomena are not characteristic of small pneumothorax except when very recent. If they persist the pneumothorax is moderately large. The collapse of lung-tissue under the pneumothorax results in increased transmission of sounds from the bronchi, if the pneumothorax is favorably situated for such transmission. On the other hand, the same collapse with its relative airlessness tends to diminish the transmission of the vesicular murmur. Finally the increasing exudate blankets all sounds. If there are several small pneumothoraces on the same side the breath-sounds over the entire lung may be reduced in loudness. The breathing over retracted lung-tissue adjacent to the pneumothorax is more or less bronchial in type. Vocal resonance is generally diminished or absent over the pneumothorax and increased over the adjacent compressed tissue. In the latter the bronchophony is modified by the resonance of the pneumothorax which exaggerates the higher-pitched elements of the sound, imparting a ringing quality. Changes in the whispered voice are among the most important and distinctive signs of small pneumothoraces. An echoing prolongation of the whispered voice is almost always a sign of the presence of a small pneumothorax. Whispered pectoriloquy of low pitch usually indicates a cavity; high-pitched whispered pectoriloquy is almost always due to partial pneumothorax. The authors believe that d'Espine's sign is evidence of a medi-

astinal or central pneumothorax which is in contact, at least in part, with the trachea.[8] If a small pneumothorax communicates with a cavity, a puff may be heard, after cough, which becomes a suction sound if the opening is somewhat constricted. Friction-sounds are not produced by a pneumothorax but may be heard in the ring of adhesions about it. The effect of a small pneumothorax is to suppress such râles as otherwise might have been heard in the adjacent lung-tissues. The absence of râles does not therefore necessarily indicate the absence of an active process. If the lung-tissue underlying the pneumothorax is not entirely collapsed râles may be present which are inconstant and give the impression of being distant and relatively "dry." The râles may have a consonating quality (in which case they are necessarily of superficial origin, or at least produced in a place which is not separated from the ear by the interposition of air-bearing lung-tissue).

From the foregoing much-condensed abstract it appears that small pneumothoraces are not to be recognized by groups of well-defined and persistent signs, but that in their study it is necessary, rather, to relate the signs obtained with the physical conditions that have produced them, the variations in the grouping of these signs affording a clue to the age and size of the pneumothorax. The researches of Barlow and Thompson have given a new meaning to localized variations in physical signs, especially as relates to high-pitched sounds, to the relative pitch of sounds and to the transmission of whisper.

The X-ray in Small Pneumothoraces.—For this study is required a thorough acquaintance with the normal lung markings. These are often obliterated in plates taken with a view to study abnormal densities. It is necessary, therefore, to adopt a special technic. The stereoscopic negatives are taken with different times of exposure. When fused in the stereoscope the dark plate predominates in the picture and gives penetration and contrast. The light plate carries the detail to the pleura in the central part and nearly to the pleura in the lateral part of the plate if the lung is normal. The chief difficulty in the diagnosis of small pneumothoraces by the roentgenograph is not in recognizing them after they have been seen but in seeing them at all. At first the diagnosis should not be made except when unmistakable separation of pleural surfaces is perceived. The chief signs of localized pneumothorax are: compressed or collapsed lung-tissue, areas of rarefaction, pleural cusps, deviation of pulmonary structures, atypical course of bronchi, atypical distribution of fibrosis, annular shadows and localized fluid levels evidently not in cavities.

[8] For d'Espine's sign see p. 232.

No one of these signs is a proof by itself of the presence of a small pneumothorax excepting actual separation of pleural surfaces.

ASTHMA

Bronchial asthma is of two types: asthma due to sensitization to proteins and the asthma of those who are insensitive to proteins. The former type is to be recognized by the history of positive sensitization tests, or by investigations which discover that condition to exist. The patients often give a family history of hay-fever or asthma. Youth is in favor of sensitization, as is also completeness of restoration of the lungs to normal between the attacks as well as suddenness of onset and disappearance of the paroxysmal dyspnea. In the asthma of the sensitized, corresponding with the rapid development of the bronchial spasm, complaint is often made of a feeling of well-marked constriction of the chest.

Because the patients of the second class do not respond to cutaneous sensitization tests it does not necessarily follow that the condition of their lungs is not in some measure due to or aggravated by the elimination, by way of the respiratory passages, of food poisons which gouty or arthritic subjects are not able to dispose of otherwise. "Gouty" bronchitis is recognizable as a nosological entity. These patients are on an average older than those of the first class. There is a history of chronic cough or of protracted periods during the cold season of the year when cough is persistent. The asthmatic attack may be preceded by an attack of bronchitis. The onset of the asthmatic paroxysm is less sudden than in the first type and the dyspnea is not so frankly expiratory. The difficulty may seem to consist rather in the expulsion of secretion than in the emptying of a lung which is overfull of air. The attack also disappears less rapidly and the lung gives evidence of bronchitic or emphysematous changes in the intervals between attacks.

It is evident from the foregoing that the typical asthmatic attack occurs in the sensitized patient. But repeated asthmatic attacks, though due to sensitization, lead eventually to emphysema and bronchitis, so that the distinction as to type is gradually effaced as the age of the patient advances.

In a typical attack of bronchial asthma dyspnea is extreme, and the peculiarity of the dyspnea is that the difficulty with the breathing is to expel rather than to inhale air. The patient opens his mouth wide and

throws his head back, thus straightening the respiratory passages, and in his unrest assumes a variety of positions all of which are such as to facilitate in some way the action of the accessory muscles of expiration. Inspiration is rapid, short and noiseless or comparatively noiseless; expiration prolonged and noisy. Sometimes a loud wheezing is audible in both phases of the respiration. The cough is dry in the first part of the attack; at a later period clumps of tenacious mucus, the "pearls," are expelled with more fluid mucous secretion. On auscultation the inspiratory sound is faint. According to Beau, inspiration may be quite without sound, as if the vocal cords were separated to their fullest extent.[9] Expiratory sound, on the contrary, is abnormally loud and much prolonged. The chest may be so filled with râles that respiratory sounds are inaudible. The râles at first are sibilant or sonorous: there is a great variety sometimes of piping, whistling and musical râles rather fine and high-pitched, as if formed in small tubes, which is undoubtedly the case. Later when the sputum becomes more fluid the characteristic asthma râles disappear and those that are present are relatively coarse and moist.

The inability to expel air from the lungs leads naturally at once to their overfilling. The lung becomes much distended, the excursions of the diaphragm are nearly or quite arrested and the breathing is almost purely costal. The position of the diaphragm is low and the pleurocostal sinus is obliterated. On percussion the note of the lung is normal or hyperresonant and low pitched. If the patient has long suffered with the disease the percussion-note owes its lowness of pitch to the fact that the lung has undergone emphysematous changes.

The dyspnea of asthma is to be distinguished from the dyspnea of obstruction and the dyspnea of heart and kidney disease. The differentiation from obstructive dyspnea is usually easy, for in the latter the dyspnea is preponderatingly inspiratory (but very exceptionally the dyspnea may be chiefly expiratory); the difficulty for the most part is to take in, not to expel, air. In heart disease the development of the dyspnea may be sudden, but both phases of the respiration are affected and the history and an examination of the heart point to the true cause.

The asthmatic attack is not always accompanied by intense dyspnea. There are cases in which it is difficult to decide whether there is a true asthmatic attack or not; that is, whether the dyspnea is due to a spasm of the bronchioles or is of a subjective nature—"pseudo-asthma." The

[9] J. H. S. Beau, "Researches on the Cause of Respiratory Sounds Perceived by Means of Auscultation," *Arch. gén. de méd., Par.*, 1834, Series II, V, quoted in "The Discoverer of the Mode of Production of Breath-Sounds," *J. Am. M. Ass., Chicago*, 1923, LXXX, 895.

distinction may sometimes be made by inspection of the costal margins. For if the whole lung is enlarged and the diaphragm is flattened the costal margins will move toward the median line during inspiration, which will not be the case if the affection is of the nature of pseudo-asthma.

BRONCHITIS

In ordinary acute tracheobronchitis it may be said that there are no physical signs so long as the affection is predominantly or exclusively a tracheitis. If the large bronchi are also involved to a material extent, râles, coarse, sometimes sonorous, extremely inconstant and irregular in distribution, may be detected. In the severer forms of bronchitis râles may be smaller and more constantly present and fever and accelerated breathing, with or without stridor, show a more serious inflammation. In general, the smaller and the more persistent the râles the more serious the case and the greater the likelihood of the presence or the development of pulmonary complications, especially, of course, bronchopneumonia.

In chronic bronchitis the important point is the determination of the cause of the bronchitis. In generalized chronic bronchitis the râles are coarse, inconstant and irregular in distribution. The same remarks as to the significance of the size of the râles heard in acute apply also to chronic bronchitis. It is certain that small râles, localized and persistent, mean more than an inflammation confined to the bronchi.

EMPHYSEMA

In emphysema, when well developed, the barrel-like, relatively immobile and enlarged chest is very characteristic. The lung is larger than it should be, hence a low stand of the diaphragm with obliteration of the pleurocostal sinus and a diminished area of cardiac dullness. The emphysematous changes are most marked at the apices and the anterior margins of the lungs. They may be confined to the anterior margins. The percussion-note is loud and clear but is not tympanitic. The lung is not only larger than normal but it is also less elastic, as the result of which changes it vibrates more slowly than the normal lung—the note of emphysema is lower than the fundamental tone of the thorax. The low pitch may also be due in part to the increased size of the thoracic cavity. The expansibility of the thorax being in some measure lost, respiration is inefficient and labored. As in asthma, the difficulty is chiefly to expel

the air; expiration is greatly prolonged. The two diseases are often associated with one another, emphysema being the chronic condition which results from often repeated asthmatic paroxysms. Since aëration is imperfect, cyanosis is often associated with the dyspnea. Cough and râles

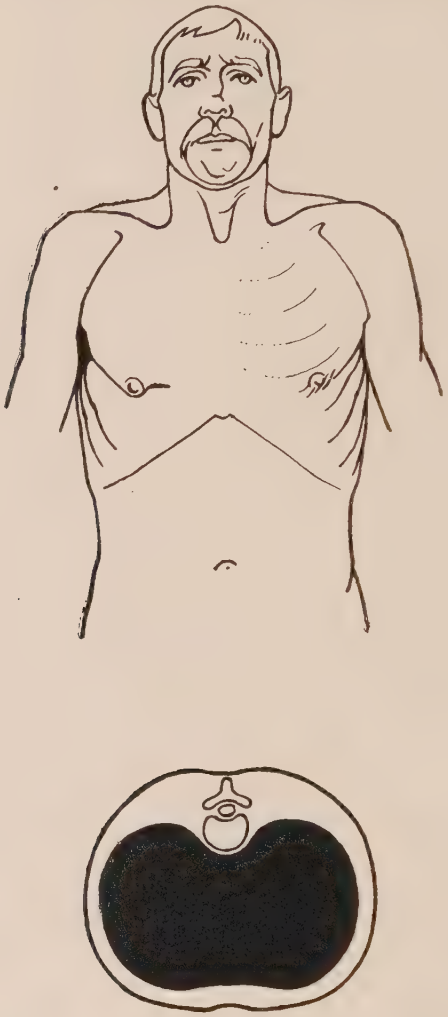


Fig. 24.—Emphysematous thorax. (Külbs, "Leitfaden der medizinischen-klinischen Propädeutik," J. Springer.)

are to be ascribed to the bronchitis which usually accompanies the emphysema. The character of the râles varies with the nature of the bronchial secretion which may be scanty and tough mucus, or, at a later period, may be mucopurulent.

As the result of the flattening of the entire dome of the diaphragm the costal margins may be drawn toward the median line in inspiration. But if the emphysema is largely confined to the anterior margins of the lungs, the flattening of the dome may be insufficient to permit this motion. The movements of the costal arch may thus furnish evidence as to the distribution of emphysematous changes. In emphysema associated with heart disease, according to Hoover, [10] it is possible to determine from the movements of the costal arch whether the dyspnea and cyanosis are due chiefly to the emphysema or to the weakness of the heart. For if inspiratory narrowing of the entire costal margin is present, it is a sign of considerable increase of the total volume of the lungs. But if inspiratory narrowing of the costal angle and retraction of the costal borders are confined to the portion of the costal margin between the angle and the eighth rib, we know that only the subcardial portion of the diaphragm, not the entire diaphragm is flattened and that, consequently, the dyspnea and cyanosis are chiefly of cardiac origin.

ABSCESS AND GANGRENE

Multiple abscesses of the lung, pyemic or bronchopneumonic by origin, are not discoverable by physical diagnosis.

Larger abscesses are single, or at most few in number. Their clinical course and consequently their accessibility to physical diagnosis depends upon their size and location. Pulmonary abscesses are to be sharply distinguished into two classes: 1. Abscesses which are capable of indefinite growth without producing marked positive tension within their walls. 2. Abscesses which in their growth produce positive tension and consequently rupture at an early period. The question as to tension depends upon the relation of the abscess to the contractility of the lung. Thus an abscess between the lobes of the lung—really an interlobar empyema—may increase to large size without positive pressure because the elasticity accommodates itself to the increasing bulk, the lung shrinking before the accumulating pus just as it does in generalized pleural effusions. A less well-known form illustrates another possibility. "Dissecting pneumonia" is recognized as a form of abscess which involves the perilobular connective tissue. A suppuration of these fibrous sheaths loosens the connections of the parenchymatous cells, leaving them intact. The acini then contract before the abscess contents *pari passu* with the increase of the latter, positive pressure being thus avoided until the

[10] "Oxford Medicine," II.

elasticity of the parts is exhausted. This form of pneumonia is common in cattle, but rare in man; possibly, however, not so rare as might be thought, for the abscess rupturing into a bronchus, the acini again expand as the contents of the sac diminish and when recovery ensues there remains little evidence of what has been an extensive suppurative process. Stokes describes a case of dissecting pneumonia in which the acini lay free in the abscess cavity like so many bunches of grapes.[11] Subpleural abscesses likewise have a relatively slight tension because of the elasticity of the lung, just as is seen in small localized pneumothoraces, though they may produce symptoms when their weight drags upon the pleura. But abscesses implicating glands or other structures at the root of the lung have little or no relief from its elasticity and, consequently, quickly acquire a positive tension and rupture while still small. The same is true of abscesses within tissues stiffened by induration or inflammatory thickening. The large closed abscess is, therefore, a superficial abscess and should be detected by physical diagnosis. The point where the abscess first becomes superficial in interlobar pleuritis depends upon the location of the original site of suppuration. Abscesses of the portion of the fissure between upper and lower lobes which is near the median line, may be detected in the interscapular region as an area of flatness on percussion and of absence of breath-sounds, with resonance above and below if the neighboring lung is normal—"suspended dullness." If situated near the axilla the abscess expands in its growth into the pleural cavity, still under little pressure if the lung is free from adhesions below for the same reason that obtained within the fissure, until it may come to rest its lower end upon the diaphragm. By physical signs such an abscess is indistinguishable from an encysted empyema of the general pleural cavity, except that its relations above to the interlobar fissure should excite suspicion. For one who has followed the growth of the abscess from its early stages with accurate physical diagnosis, the course of development should point unmistakably to its origin and nature. Aspiration of such an abscess does not differ from that of ordinary empyema, but incision would often not be followed by a gush of pus but by the production of a pneumothorax, the abscess following the contracting lung without rupturing under the violent change of the conditions. Cure of even so large an abscess may result from rupture into a bronchus and after rupture and evacuation it may disappear so far as physical signs are concerned, the neighboring parenchyma of the lung, which has been merely collapsed, expanding practically to its normal

[11] William Stokes, "Diseases of the Chest," 1839.

limits. Large abscesses follow lobar pneumonia, as a rule, and especially pneumonia after influenza.

Pulmonary tuberculosis is not characterized by large abscesses. When the tuberculous abscess is a "cold" abscess—that is, due to the tubercle bacillus alone—it amounts simply to a liquefaction of caseations. Sometimes, however, an abscess pursues a more acute course in pulmonary tuberculosis, probably because of infection with pyogenic organisms, and ruptures early. Such small abscesses usually give no physical signs decipherable from those of the surrounding, usually pathologically altered, lung.

Gangrene is the more likely to occur in the lungs the more septic the infection. It follows the abscesses of septic bronchopneumonias or may appear coincidently with abscesses. In such lesions the gangrene is not susceptible of identification by physical signs. And, in general, gangrene often presents itself upon a background of tissues which have already undergone profound pathological changes, so that the addition of gangrene brings little to identify it except the unmistakable fetor. However, at times larger areas become gangrenous, usually perhaps as the terminal result of infarction. This occurs sometimes in far advanced cases of pulmonary tuberculosis with none of the usual signs of thrombosis and embolism, not even hemoptysis. The change from the variety of physical signs, so abundantly present in the involved region previously, to silence occasionally broken by distant and faint breath-sounds and coarse râles conveyed through the dead tissue, is striking; the gangrenous region is, of course, motionless and dull on percussion.

ATELECTASIS

The term "atelectasis" signifies an airless condition of the lung-parenchyma due to collapse of the air-cells. It is a normal condition during fetal life. In later life the return of the lungs to the fetal state is only possible under one of two conditions: The afferent air-tubes must be completely blocked, or if the tubes are free the lung-cells must be under pressure. Collapse alone does not empty the cells of air. The disappearance of the alveolar air is effected by absorption through the blood of the capillaries. Absorption alone does not remove all of the air of a closed space, for it ceases when the tension of the air to be absorbed equals that of the gas of the fluid. However, the elasticity of the lung, while it is not strong enough to collapse the air-cells completely, nevertheless

suffices to contract the cell walls as the absorption progresses so that the tension is not lowered and in the end the occluded tract becomes completely airless. In a region of compression the pressure aids the elasticity in reducing the size of the air-cells and at the same time prevents inspiratory expansion with renewal of the air, so that the same end result is reached.

Occlusion of a bronchus or bronchiole is a common cause of atelectasis in pulmonary tuberculosis, but the condition is not usually recognized during life. The tuberculous lymphangitis which is a feature of even the earliest and smallest tuberculous lesions involves the lymphatics of both blood-vessels and air-tubes. The walls of the blood-vessels are highly resistant to the invasion of the tuberculous process, those of bronchi and bronchioles are much less so. Owing to the low resistance of the bronchus a tubercle involving its coats and projecting more or less into its lumen is of common occurrence. If the protrusion is sufficient to close the air-tube completely atelectasis develops in the part of the lung supplied by the tube. The condition is not usually appreciable clinically on account of the smallness of the lesion. Only the small air-tubes are occluded, in this manner. When larger bronchi are blocked the occlusion is more likely to be due to the direct involvement of the tube in the processes of caseation, and the tuberculosis of the tributary air-cells is already so far advanced that the final result is usually pus-filled ramifications of lesser tubes amid tissues already caseous, or at least so nearly airless from long participation in the tuberculous process that the rôle of atelectatic collapse is insignificant.

Atelectasis from compression occurs chiefly in connection with pleural exudation. The lung that lies beneath the level of the exudate is compressed more or less by hydrostatic pressure, or in larger accumulations, by the positive pressure of the effusion. In both occlusion and compression there is a well-marked pathological process leading to lung collapse and ultimately to atelectasis, irremediable usually in the first, generally temporary in the second.

The excuse for these pathological details in a treatise devoted to physical diagnosis is that the writer believes it to be well to call attention to the facts which show that true atelectasis is not acquired by a relative and temporary disuse of any portion of the lung. In fact, in his judgment the readiness with which "atelectatic" râles and "unfolding" inspiration of the bases and apices of the lungs are assumed as explanation of adventitious sounds in these regions leads too often to erroneous assumptions, not only as to the pathological condition of the lung in question, but also as to the frequency of atelectasis. In order to make

these points clear it will be necessary to discuss them at some length.

A difference of opinion prevails as to the activity of respiratory function of the apex. According to some the apex is imperfectly aërated. There can be no doubt, however, that the upper portions of the lungs bear the brunt of cough and that they are expanded with the remainder of the lung in the deep inspirations that follow a coughing fit. But when the breathing is chiefly diaphragmatic—and, naturally, most conspicuously in persons of a sedentary life—the first full breath or two after quietude is apt to be accompanied by rather dry râles. These are small râles, but not as fine as the crepitant râle. It is, therefore, impossible that they can represent the unfolding of the alveoli from a state of atelectasis. Undoubtedly the little-used apex becomes congested in persons predisposed to such congestions and hence easily somewhat "soggy." The slight and therefore presumably sticky moisture in the small air-tubes produces râles when first disturbed which quickly disappear, but their disappearance does not mean that the sounds had been produced by the separation of surfaces formerly in contact, since the same phenomenon —that is, the presence of fugitive râles—often occurs elsewhere in the lung where there can be no suspicion of atelectasis. (See p. 208.) At the same time that the adventitious sounds are heard during the more effective expansion of the lung, the apex may be seen through the fluoroscope to become lighter, that is, less congested. Such congestion may occur in cases which present signs of an indurative process; it is also seen in cases in which the only lesion of the apex is pleural adhesions. Congestion from relative disuse of an apex crippled by the fibrosis and the pleural adhesions of obsolete, usually tuberculous, processes accounts satisfactorily for the presence of the so-called "atelectatic" râles of the apex.

The "Atelectatic" Râles of the Costopleural Sinus.—If a person whose breathing has been shallow is incited to respirations of somewhat greater depth, a few sounds like moist râles may be heard at the base of the lung, as the lung, expanding, is drawn into the costopleural sinus. These sounds disappear, as a rule, after a few breaths. The phenomenon is not often noted in the systematic exploration of the chest because the examination rarely begins with the bases, and the sounds have disappeared before the place of their production is reached in the course of the examination. Hence they are usually discovered in examinations of patients who have pain in that region or who for other reasons may call especial attention to that part of the lung. Finding the "râles" in the part complained of, it is natural for the examiner to infer that they are a part of a morbid process of some kind. When, as is more commonly the case, no evidence of actual disease is to be made out, one of two

conclusions is usually drawn; if there are no other symptoms, that the sounds are a sign of atelectasis; if the patient chances to complain of pain in that region, that there is a dry pleurisy. We hear often of "airless and soggy bases" and of the frequency of pleuritis at the base of the lung. But the fact is that airlessness does not occur in this, the most powerfully breathing part of the lung, in the absence of pleural effusion or of inflammatory infiltration, and that pleurisy of the complemental space is not more, but less common than pleurisy of the lung higher up, as the writer has convinced himself by extended observations at autopsy.

In order to understand the laws which govern the production of the sounds in question, reference must be made to the physiology of respiration as concerns the pleurocostal sinus. (See p. 120.) The inner wall of the costopleural sinus—or the complemental space, as it is sometimes called —is formed by that portion of the diaphragmatic pleura which comes into contact with the chest wall as the diaphragm relaxes and follows the lung upward in its expiratory ascent. At the bottom of the sac the pleura is reflected from the ribs to cover the dome of the diaphragm. When this point, the costodiaphragmatic angle, is reached in inspiration the fullest expansion downwards of the lateral surfaces of the lungs has been attained. The diaphragm, as it contracts and descends in inspiration, separates the diaphragmatic from the costal pleura with which it has been in contact. The surfaces being moist, their separation is attended by sounds that characterize the peeling off of one moist and slightly tacky surface from another. These sounds, the marginal sounds, are usually audible on auscultation. They resemble moist râles if the separation is made slowly; the sounds are drier if the separation is more forcible; when the breathing is very rapid they are not to be made out at all. In most cases the sounds disappear after a few breaths, but in well-trained subjects they are producible indefinitely. The marginal sounds are heard better on the affected side in tuberculosis of the upper lung than in the normal lung of the other side, where they are often absent or less well marked. For this reason they have been considered to be a sign of tuberculosis and by some have been held to be evidence that apical tuberculous lesions are accompanied by basal lesions. Why is it that these sounds disappear so readily in the sick, or in the poor breather, are sometimes conspicuously present in tuberculous disease yet continue indefinitely in the vigorous breather? The explanation is simple and if understood will do away with many misconceptions.

As for the disappearance of marginal sounds, the lung is not a perfectly elastic organ. Hence if inspiration is forcible but expiration remains passive the lung will become temporarily distended. It will then

occupy the pleurocostal sinus, either entirely or to such an extent that the descent of the diaphragm is not accompanied by sound. The proof of this is afforded by the fact that if the trained breather is required to take a number (six to ten) of deep inspirations followed by shallow expirations, marginal sounds cease to be heard, however deep the following inspiration may be. But if he reverses the process and deflates the distended lung by the same number of respirations, in which expiration is forcible and inspiration shallow, the marginal sounds return. It is only when the costopleural sinus is empty at the beginning of inspiration that the separation of the walls of the sac will be made through a sufficient range to produce marginal sounds. As has already been remarked, the untrained breather always leaves expiration to the passive recoil of the parts that have been expanded by the previous inspiration. If inspiration is at all deepened in this type of breathing the result is quickly distention of the lung and disappearance of marginal sounds.

A similar explanation accounts for the behavior of marginal sounds in unilateral pulmonary tuberculosis. The affected side is impaired in its elasticity by the presence of the tuberculous lesion. For this reason it expands less in ordinary quiet respiration than the sound lung which does the greater part of the work. But the lower lobe being by supposition normal, the diseased lung is still able to expand to fill the sinus when called upon for especial inspiratory effort, even to the extent of a slight increase in the depth of inspiration, and as it enters the empty sinus marginal sounds are readily produced. On the other hand, the healthy lung, having been acting to some extent vicariously for the diseased lung, is somewhat distended; its margins occupy a considerable part of the depth of the sinus before inspiration begins; consequently marginal sounds are not heard or are inconspicuous.[12]

If the lung is abnormally, or unusually, expanded, as in emphysema or temporarily after mountain climbing or other severe and prolonged muscular exertion, there is, for the time, no costopleural sinus. And, in general, the depth of the sinus depends upon the volume, for the moment, of the resting lung. The performance of the lung in inspiration depends upon its ability to expand. That lung will expand best in inspiration which has been most effectively emptied in the preceding expiration. The trained breather produces marginal sounds for as long a time as may be desired because his expiration is as forcible as his inspiration, so that the lung is not permitted to become distended.

Another proof of the correctness of the foregoing explanation is af-

[12] For a more full discussion of this subject see Bushnell, "Marginal Sounds in the Diagnosis of Pulmonary Tuberculosis," *Med. Rec., N. Y.,* Dec. 21, 1912, LXXXII.

forded by the phenomenon known as "reversible crepitations." If a subject lies horizontally on one side very pronounced marginal sounds are heard on deep breathing over the lower side; they are absent on the upper side. But if he turns over to lie upon the other side, the marginal sounds are heard over the side that is now the lower side and again are inaudible on the upper side; the crepitations are "reversed." The phenomenon is due to the sinking of the abdominal contents to the dependent side. In so doing they push up the diaphragm on that side so that in expiration it occupies a higher position than when the subject was erect. But on the upper side the traction of the abdominal viscera diminishes or abolishes the upward pressure usually exerted by the contents of the abdomen upon the diaphragm; that pressure may be said to have become negative. The lung of the upper side is, therefore, free to expand and may fill the pleurocostal sinus more completely than it does in any other position of the body. But there being little or no upward pressure from the abdominal contents to aid its recession in expiration, it remains more or less distended, occupying the sinus to such an extent that marginal sounds are not produced. On the other side, the lung being pushed up farther than usual, the sinus is completely empty; in fact is deeper than usual because the diaphragm is in apposition with the ribs at a higher level. Hence, though when it descends it must act against a greater resistance, a vigorous inspiration causes a descent through a greater distance. Marginal sounds are therefore produced with especial distinctness.

Through the fluoroscope the diaphragm of the lower side is seen to occupy a higher position than usual in expiration. Its descent is vigorous during inspiration and through a wide range. On the upper side, however, the descent of the diaphragm during inspiration is slight and its movements are of an undulatory character, as if communicated to this half of the organ from the other. This very interesting behavior of the diaphragm illustrates well the complete dependence of the diaphragm-half upon the lung with which it is in contact. On the upper side there can be little or no production of marginal sounds, for the diaphragm, being already more or less flattened, can not descend in inspiration to any material extent. (See p. 133.)

When by powerful contractions of the expiratory muscles, the intestines and other abdominal viscera are pushed back into their usual position, breathing again becomes of the normal type and a strong breather may even produce marginal sounds at the base of the lung of the superior side.

The student should practice assiduously upon the healthy subject, per-

cussing out the lung borders and the level of the lung bases in the extremes of inspiration and of expiration, both in the erect position and in lateral recumbency, and studying the relative production of marginal sounds on both sides while the subject is lying in the horizontal-lateral position.

No diagnosis of disease unsupported by other unmistakable evidence should be made on the ground of what are taken to be râles or friction-sounds heard over the base of the lungs in inspiration, until marginal sounds have been excluded.

Marginal sounds are strictly confined to the pleurocostal sinus; disease of the lower lobe is rarely limited to the extreme margin of the lung. If the lower lobe is unmistakably involved in a morbid process, the distinction between marginal sounds and true râles becomes difficult; but whether marginal sounds are present or not under these circumstances is a relatively unimportant question.

Pleural adhesions which sensibly restrain the lung movements usually prevent the production of marginal sounds. The presence of these sounds, like that of the shadow of Litten, is proof of normal mobility of the diaphragm and, therefore, of the probable absence of pleural adhesions at the base of the lung and also of the absence of well-marked local pathological increase of volume in the vicinity of the base of the lungs whether above or below the diaphragm.

PULMONARY EDEMA

In this affection there is an escape of serum from the blood-vessels into the air-cells and air-tubes of the lung. There is no sharp line of distinction between congestion of the lung and edema and much of what is called hypostatic congestion, for example, under the definition is really hypostatic edema. Edema varies as to amount of moisture from a small amount of fluid to the entire or nearly entire filling of the lung. In its more marked forms edema is an exceedingly common finding at autopsy. It is not an independent disease but might be defined as the pulmonary expression of a weak heart and an obstructed circulation. The gravity and importance of edema are that which attaches to the morbid condition that caused it.

The physical signs of edema are of importance in the distinction between this condition and other affections which cause difficulty of breathing. Pulmonary congestion gives few physical signs. The percussion-note is higher pitched and dull from diminution in the vibratility of the lung,

and moist râles are generally to be heard. If the congestion passes into pneumonia the signs will be more or less typical of that inflammation, according to the amount and the completeness of the hepatization. The diagnosis of this complication is, however, generally difficult on account of its atypical character. The acuter forms of edema are more threatening to life and at the same time easier of identification. According to Gerhardt, if fluid is injected into a normally inflated lung after death, the note of the lung becomes tympanitic. Tympany under such conditions always depends upon the presence of both air and liquid in the alveoli. When the air-cells are completely filled with serum or water the percussion-note is dull or flat. Tympanitic resonance means of course that the lung or part of the lung that is edematous vibrates as a whole by virtue of a relaxation from the normal lung tension. It would hardly be expected that a lung thus weighted with fluid would have the degree of vibratility that the facts seem to demand; it is probable that the amount of fluid in the individual cells is small at this stage. When the heart fails rapidly there may be severe dyspnea and a sense of oppression without definite physical signs in the lung, with the possible exception of a few râles. This condition is sometimes called "cardiac asthma"; the term however is often quite loosely applied. The condition is distinguished from edema by the absence of serous effusion with its characteristically numerous moist râles. Bronchial asthma is not likely to be mistaken for edema, however intense the dyspnea. If doubt should exist it is dispelled by examination for râles. The sibilant and piping râles of asthma stand at the opposite extreme from those of edema. The nature of the various sounds of the asthmatic chest and the character of the mucous pellets which are expelled show clearly enough that the secretion of asthma is of the most viscid mucus. But in edema the râles are due to bubblings in serum, not mucus. They are "serous râles" and their character is what would be expected in a fluid practically devoid of viscosity. Emphysema, as well as congestion, may be present with edema. The apices and especially the edges of the lungs may be emphysematous when the remainder of the lung is edematous. If it were true that emphysema is characterized by tympanitic resonance, the only means of distinction between emphysema and edema by physical signs would be the abundant râles of the latter. But the resonance of emphysema is not tympanitic. The relatively inelastic and relatively motionless lung of emphysema does not vibrate freely. At best, the tympany of emphysema is a dull tympany, the "Schachtel," or bandbox tone; that is, a noise poor in deep tones because of the increased tension of the lung and of

the chest wall. The frequent coincidence of emphysema with edema probably accounts for the observations in which the note of emphysema is said to have been tympanitic.

FIBROID PHTHISIS OR CIRRHOSIS OF THE LUNG

The reaction of the lung to persistent irritation and to chronic infections is in general, as elsewhere in the body, by the formation of fibrous tissue. A great variety of causes may lead to such an increase of fibrous tissue which, according to the dissemination of the irritant, may be diffuse or localized. Massive induration in non-tuberculous disease is usually the last stage of an organizing lobar pneumonia. In the bronchopneumonic type the alveolar exudate becomes organized in the same way but the foci are more scattered. They may, however, fuse so as to lead to indurations as massive as those of the lobar type. In a third type, the pleurogenous, the inflammatory changes in the lung originate after collapse due to accumulations of some kind in the pleural cavity.

The indurated lung contracts ultimately to an extreme degree and the contraction results in great displacement of the heart toward the lesion, and in elevation of the diaphragm with relative immobility of the lung of the affected side. Sometimes the indurative changes involve the whole or the greater part of both lungs, as in pneumonoconiosis, a type of induration due to dust, especially to the dust of granite. Irritation from the inhalation of such dust, if long continued, produces numerous small foci of inflammation, much resembling miliary tubercles in the x-ray, which are disseminated more or less universally throughout the lungs and lead to a generalized induration and, ultimately, to death. Such cases are very often complicated by tuberculosis and the question has been much debated which is the factor chiefly responsible for the fatal issue, tuberculosis or pneumonoconiosis. On the principle that all cases with a positive sputum are counted as tuberculous, such deaths are generally ascribed to tuberculosis; but at present investigators appear to incline to the opinion that the chief cause of death is the injury to heart and lungs from the contraction of the fibrous tissue and that the tuberculosis, engrafted as it were upon the fibrosis, is incapable of rapid or typical extension and is therefore relatively benign (as it so often is in aging persons). The diagnostic signs of indurated tissue are the same always irrespective of the cause of the induration—dullness on percussion, harsh breath-sounds generally bronchovesicular rather than purely bronchial, a variable, but

generally increased transmission of voice and whisper, and râles which usually proceed preponderatingly from the larger bronchi and are, therefore, coarse or of medium size. As a rule the quality of the râles is "viscid," the cough is severe and the sputum is raised with some difficulty. But in bronchiectasis, which often complicates pneumonoconiosis, or more rarely in abscess, the sputum may be abundant, purulent and foul-smelling and perhaps raised with comparative ease.

In diffuse fibrous induration, the affection being bilateral, displacement of the heart may not be marked. When one side is distinctly more involved the displacement will be to the side of the greater involvement. But pleural adhesions, here as elsewhere, may lead to apparent exceptions to the laws of displacement of the mediastinum.

In diffuse fibrosis in its early stage the percussion-note is impaired by the general diminution of the vibratility of the lungs. The individual lesions do not give localized dull areas of percussion. But as the disease advances larger scattered indurations of a bronchopneumonic type may develop which, if superficial, are accessible to percussion. The three types of induration are rarely to be made out with distinctness, even at autopsy: the clinical picture as revealed by physical diagnosis is usually that of irregularly distributed masses of induration.

BRONCHIECTASIS

So far as physical signs are concerned, a bronchiectasis is a cavity. Bronchiectatic dilatations, which are found much more rarely clinically than at autopsy, are usually associated with indurative changes; in fact the production of the cavity is probably largely due to the contraction of fibrous tissue. In this case the physical signs are those of indurative inflammation or of fibrosis, plus those of cavity. Bronchiectasis is most common in tuberculosis, in which disease the differentiation from cavity due to loss of parenchyma is difficult and not important. If bronchiectasis occurs as the result of indurative pneumonias of the lower lobes which follow acute infections or are due to aspiration of foreign bodies, embolism and the like and pulmonary tuberculosis is absent or confined to the apex, the bronchiectasis in all probability is not tuberculous. No doubt, as in fibroid phthisis, the presence of a sputum positive for the tubercle bacillus often leads to a diagnosis of pulmonary tuberculosis as the principal disease that is not justified.

AFFECTIONS OF THE MEDIASTINUM

The posterior mediastinum is far removed from the surface of the back by the interposition of the spinal column. Only very large abscesses or tumors in this region give evidence of their presence by changes in the percussion-note; and such evidence, in the form of dullness over the lung neighboring upon the spine, proceeds rather from the compression of the lung than directly from the mediastinal abscess or growth itself. However, it is claimed that with very light percussion dullness may be made out over the spinous processes. D'Espine's sign has been discussed elsewhere.

Anterior mediastinitis may occur in the form of inflammatory edema in lobar pneumonia, the infection proceeding by way of the lymphatics. Diffuse infiltration can hardly be detected, but a large abscess may so damp the resonance of the sternum and of the regions of the lung adjacent to it on either side as to produce dullness on percussion. The same is of course true of tumors involving the mediastinum directly and of glands enlarged by tuberculous inflammation or cancerous metastasis. Dermoid cysts sometimes are found in the anterior mediastinum.

True râles, if heard over the sternum, are not of course mediastinal but pulmonary sounds, since for their production the presence of air is necessary. Such râles, therefore, mean nothing as to the presence of a mediastinitis. It is altogether probable that the sounds regarded as râles and as signs of a mediastinitis are practically always sternal sounds.

It is extremely important to note the remarks on the subject of sternal sounds in Chapter III (see p. 112) to become familiar with the sounds which may normally be elicited over the sternum before attempting the diagnosis of affections of the mediastinum.

PULMONARY SYPHILIS

Syphilis of the lung is rarely recognizable clinically. Histologically it is differentiated with difficulty from tuberculosis. Bronchial catarrh may be one of the manifestations of the secondary stage of syphilis. Gumma is a late lesion, belonging to the tertiary stage. Gummata are generally small and may be found in any part of the lung. They are more often deeply situated than superficial. A diffuse fibrosis with or without gumma may develop as a result of the syphilitic process and is frequently

accompanied by bronchiectasis. Stenosis of trachea or bronchus may be caused by syphilitic ulceration or by gumma. This stenosis may lead to inflammation and induration of parts of the lung below the lesion which are due to the obstruction of the air-passages rather than to syphilis directly. Deep gummata give no physical signs. The physical signs of syphilis of the lung are in general indistinguishable from those of tuberculosis, but deep-seated lesions involving solely or chiefly the lower lobes or scattered irregularly through the lung should excite a suspicion of syphilis. Tuberculosis and syphilis sometimes are present contemporaneously in the same individual. If a patient with active tuberculosis acquires syphilis the outlook is unfavorable. In fact the prognosis is not good if syphilitic has preceded tuberculous infection, provided that both diseases are recent and active. On the other hand tuberculosis developing in subjects with old fibrous tertiary syphilitic lesions appears to run a relatively benign course. This is probably especially true if fibrosis from syphilis has involved the lungs to any considerable extent. The situation would then be comparable to that which arises when tuberculosis is engrafted upon a lung fibrotic from pneumonoconiosis.

MALIGNANT DISEASE OF THE LUNG

Both carcinoma and sarcoma attack the lung. Cancer of the lung is a rare disease; sarcoma is still rarer. Malignant disease may develop in the ulcerations of the bronchi which are due to syphilis or tuberculosis. Cancer of the bronchi is likely to result in partial or entire occlusion. In the former case the phenomena of bronchial stenosis develop; in the latter the occlusion leads to atelectasis of the portion of the lung supplied by the occluded bronchus.

In pulmonary tumors the deeply situated growths give at first the indications which arise from the presence of any solid body within the lung—unequal expansion of the two lungs with possibly the dislocation of the heart toward the affected side and pressure signs which vary with the location of the tumor. The x-ray shows a dense opacity.

The root of the lung is sometimes the seat of cancer, either primary in that location or metastatic in the hilus glands. The extension of cancer is largely by the lymphatics. In especially malignant forms the cancer extends rapidly peribronchially through the lymphatics from the hilus in a manner that recalls peribronchial tuberculosis, but with the difference that the extension of cancer is by growth of solid tissue rather than as a lymphangitis.

Cancer may invade the lung by way of the bloodstream as metastases from tumors situated in other organs.

Pain and dyspnea are frequent symptoms of pulmonary malignant disease. There is generally cough with at first little or no expectoration. Hemoptyses are not uncommon; the sputum may be bloodstreaked and at times has the appearance of currant jelly. This has been regarded as a diagnostic sign of cancer but is not limited to it.

The physical signs vary greatly according to the site and size of the tumor. Since pulmonary tuberculosis is a common disease and cancer comparatively rare, it is to be expected that the diagnosis of tuberculosis will frequently be made in cases really of malignant disease. In many respects the physical signs of the two diseases are very similar and the more so when secondary necrosis of the cancerous growth has led to the formation of cavity. However, there is the cardinal difference that pressure symptoms are common in cancer and almost unknown in pulmonary tuberculosis. As the result of pressure which renders lung-tissue quite airless, bronchial breathing is sometimes heard in a perfection not equalled in pulmonary tuberculosis. The only absolute proof of the presence of malignant disease is afforded by the microscopic examination of fragments that may be expectorated from time to time.

INFLUENZA AND STREPTOCOCCIC AFFECTIONS

Influenza owes its importance as a cause of death chiefly to the fact that it prepares the way for other infectious microörganisms, especially the pneumococcus and the streptococcus. Its influence is also traceable in increase of acuity of the infections that complicate it. The streptococcus might almost be said to have played a new rôle during and since the war in the causation of bronchopneumonias of a more or less chronic nature the course of which greatly resembles the tuberculous bronchopneumonias, even to the occurrence of hemoptysis, the only ground remaining for the distinction of the streptococcus from the tuberculous infection being the negative sputum of the former as respects the tubercle bacillus. On account of this imitation it was not easy, in the army camps during the war, to make a diagnosis of active tuberculous conditions without a period of observation and repeated examinations of the sputum. The streptococcus may likewise cause a chronic bronchitis with slight fever and abundant mucopurulent expectoration—a fact which has deprived of most of its weight the former dictum that bronchitis in young persons means as a rule tuberculosis. Absence of local foci and a sputum

negative for the tubercle bacillus but containing streptococci either in pure culture, or, later, as the predominant microörganism, are the points which should generally establish the diagnosis in favor of streptococcus infection.

The question whether influenza affects unfavorably the course of pre-existing pulmonary tuberculosis or reactivates previously quiescent tuberculous infections has been considerably discussed and writers do not agree as to its solution. As a cause of impairment of the general health influenza may and probably does turn the scale at times in favor of the reactivation of tuberculosis. But patients with already existing tuberculous disease, experience shows, are not more, but less, seriously attacked by influenza than the average person of the same age. There seems to be no doubt that, in so far as a lung is affected by fibrotic changes, it becomes more, not less, resistent as respects the reception and the acuity of development of new infectious diseases. The course of cases of pulmonary tuberculosis in which bronchovesicular breathing and more or less dullness on percussion have shown the existence of some degree of fibrosis are not, therefore, likely to be materially affected for the worse by an attack of influenza.

"GASSING"

Exposure to certain war gases, notably "mustard gas," leads to injury of the bronchial mucous membrane which varies in severity, according to the degree and length of exposure, from temporary irritation to severest bronchitis. The diagnosis of the acute bronchitis of recent cases is based as to etiology upon the history of exposure to gas and presents no special difficulties. The severer cases, however, may go on to develop a chronic bronchitis which it is not easy to distinguish from chronic pulmonary tuberculosis of the peribronchial type. The signs and the x-ray picture are practically the same in the two diseases, except that the lower lobe bronchi are more preponderatingly affected in bronchitis from gassing than is usual in pulmonary tuberculosis. Hemorrhage, in which blood is lost to an appreciable extent, is characteristic of tuberculosis, but blood-streaked sputum is not uncommon in bronchitis from chemical injury. The cardinal diagnostic point is, of course, the presence or absence (after repeated examinations of the sputum) of the tubercle bacillus.

Diffuse fibrosis of the apices and contraction of Krönig's isthmus point to a tuberculous process of considerable duration, but do not decide whether or not that process continues to be active elsewhere in the lung. If the sputum is found positive for the tubercle bacillus the problem is to determine whether the active disease is due chiefly to the old injury of the

bronchi or to tuberculosis. Fever, progressive loss of weight and hemoptysis are usually to be regarded as indicative of a progressive tuberculosis. It does not appear that war-gassing tends to cause tuberculous infection to become tuberculous disease. The gassed patients who prove to be also tuberculous come largely from families known or suspected to be tuberculous,[13] which fact shows that, in all probability, they brought their tuberculosis with them into the army. It is interesting to note that Cole states that "the gassed patient who later develops tuberculosis usually runs a less severe course, with less tendency to serious complications, than the non-gassed individual." [14] This fact is to be interpreted as in the tuberculosis which occurs in syphilis and pneumonoconiosis; in all conditions which produce a pulmonary fibrosis an engrafted tuberculosis tends to pursue an especially chronic and relatively benign course.

HYDATID CYSTS

Cysts may be found in any part of the lung but are more frequent on the right side and in the right lower lobe. Cysts vary greatly in size and may long remain latent. Deep small cysts are not accessible to physical diagnosis. A large unruptured cyst which approaches the pleural surface of the lung gives the signs of an airless body—flatness or marked dullness, absence of vocal fremitus and diminished or absent transmission of breath-sounds, voice and whisper. The dull area is more or less circular in form and the transition at the edges to lung-resonance is rather abrupt. Hemoptysis may occur previous to rupture and there may be dyspnea and pain. When the pleura is involved the pain may be severe. After the sac has broken the signs are those of cavity, often surrounded by a pneumonic area; or if the pleura is implicated there may be pleurisy with effusion and sometimes pneumothorax. If pus is coughed up from an inflamed and ruptured cyst the affection may be confounded with amebic abscess. Subdiaphragmatic cysts developing immediately below the diaphragm or in the liver, spleen or kidney may push up the diaphragm and compress, without actually invading, the lung. In these cases the diaphragm is very high and dullness, progressively diminishing upward, may be made out in the lung above it.

TYPHOID FEVER

Typhoid fever may be mistaken for tuberculosis if it is of the type in which there is considerable pulmonary congestion manifested by numerous

[13] H. H. Cole, "A Clinical Study of the Gassed Ex-soldier with Especial Reference to Pulmonary Tuberculosis," *Am. Rev. of Tuber.*, 1923, VII, 230.

[14] *Loc. cit.*

moist râles. However, in this type the usual signs and symptoms of typhoid are as a rule well marked, the râles are disseminated through the lungs and unless bronchopneumonia develops there is no semblance of the local foci characteristic of pulmonary tuberculosis.

Not very infrequently febrile pulmonary tuberculosis is for a time taken to be typhoid fever. There is little excuse for this if the examiner has been trained in the physical diagnosis of lung disease.

It is a more difficult problem to distinguish typhoid fever in which the diagnosis has been made by exclusion, and the disease known in the West as "mountain fever" in which there are often no symptoms except fever and weakness, from the early stages of miliary tuberculosis; in fact, the diagnosis can only be made clinically after a sufficient interval of time has elapsed to permit the miliary tuberculosis, if present, to declare itself. The diagnostic abilities of the physician come into play in detecting the earliest indications of tuberculosis. Usually mountain fever occurs in the summer or autumn and many persons fall ill of it at about the same time; a fact which goes far to indicate the probabilities in the given case.

PULMONARY ACTINOMYCOSIS

Actinomycosis may be primary in the lung or reach it by extension from other parts—the neck and mediastinum, sometimes the esophagus or from the abdomen through the diaphragm. Metastases may also occur by invasion of the bloodstream. In pulmonary actinomycosis bronchitis is almost always present. The parenchymatous lesions are those of a bronchopneumonia and of the sequels of bronchopneumonia—induration, bronchiectasis and multiple abscesses. The disease is usually unilateral. Implication of the upper lobes occurs but is less common than that of the lower lobes. In early pulmonary metastasis miliary nodules may be scattered through the lungs or larger wedge-shaped areas may occur subpleurally. The pleura is implicated as a rule, the pleuritis usually terminating in adhesions. The physical signs of actinomycosis are indistinguishable from those of tuberculosis. The localization of the foci is not generally that characteristic of pulmonary tuberculosis. The location of the disease elsewhere in the body and its mode of extension should lead to the probable exclusion of tuberculosis. The diagnosis is made by the discovery of the characteristic granules by microscopic examination of the sputum, which is more distinctly purulent than that of tuberculosis.

Other parasitic fungi, the streptothrix, the aspergillus and the organism

of blastomycosis, all may infect the lungs and produce lesions indistinguishable from those of tuberculosis, the diagnosis requiring to be made by the microscope. In blastomycosis there are usually cutaneous lesions, in the pus from which the capsules of the microörganism may be detected.

CHAPTER V

PULMONARY TUBERCULOSIS

Pathology.—For the correct diagnosis and prognosis of the lung lesions, and especially of the chronic lung lesions, of tuberculosis it is necessary to know something of the pathology of these lesions.

Tuberculosis attacking an entirely defenseless organism develops rapidly and fatally. No human organism is entirely without means of resistance, but in many cases the resistance is so ineffectual that it may be ignored, and the progress of the disease and its mode of universal extension may be said to depend upon the physical conditions which favor the dissemination of the tubercle bacilli throughout the body and upon the rate of their multiplication. The study of such a hopeless case is comparatively uninteresting and unimportant, for it amounts simply to the study of the unopposed progress of the infectious agent.

On the other hand, when the first tuberculous infection has been produced by a small number of tubercle bacilli and the natural resistance of the individual is good, the anatomical evidences of the infection are not discovered without special search. The first tuberculous lesion of the parenchyma of the lung in this type is insignificant in size, benign in character and when first examined is almost always found to be approaching arrest or already obsolete. Even at an early age such a lesion presents evidence of the acquired resistance to the tubercle bacillus which results from a favorable reaction to infection, or, at all events, if truly primary, has a pathological histology which strongly resembles that of lesions known to be secondary in point of time. The fact that a case of pulmonary tuberculosis is chronic in its course leads us necessarily to assume that there exists an acquired resistance or relative immunity to the tubercle bacillus. In such cases the tubercle bacillus may be of the same character as those which fatally infect other less fortunate individuals. The difference between the result in the two classes of cases depends upon the initial dosage with tubercle bacilli and the relative natural and acquired resistance of the human organisms concerned. In the resistant class we study not so much the infectious agent as the resistance of the patient to it—we study the patient, not the tubercle bacillus.

To understand, then, the tuberculous process we must learn the laws of

the resistance of the more or less immune organism, how defenses are prepared, what conditions favor defense, what attack. In short, we must connect our physical diagnosis as closely as possible with the various modes of development of tuberculous disease which progresses, if it progresses at all, against a more or less successful, but varying resistance.

In fatal cases in infancy what is probably a primary tuberculous infection in the lung appears as large caseous foci, with or without excavation, and much enlarged and caseous or suppurating hilus or tracheobronchial glands. The most conspicuous features of the disease may be found in the lungs, but the infection is really a generalized one and death usually results from meningitis or from miliary tuberculosis.

The frequency of small calcified foci in the superficial portions of the lungs with more or less extensive calcifications in the corresponding hilus gland has long been recognized. It has remained for Ernst Ranke to include with the histological examination of the deep and superficial lesions a systematic study of the lymph channels that connect them. The following description is based upon his work.[1]

The tuberculous focus of the hilus gland, which in the obsolete lesion is completely calcified, is generally considerably larger than the, likewise usually calcified, parenchymatous focus or foci. Bands of connective tissue arise in the hilus region in close proximity to the affected gland and pass toward the superficial lung. These bands are closely related to the blood-vessels, appearing as a thickening of the adventitia. At first they are only in so far connected with the bronchi as the vessel and the bronchus normally accompany one another. But the bronchus is soon implicated in the inflammatory proliferation of connective tissue which often extends as far as the bronchial mucous membrane and in that case the bronchus shows a certain amount of chronic inflammation. While the proliferative processes seem to advance by preference perivascularly, the inner coats of the vessels manifest a high resistance; the resistance of the bronchi, on the other hand, is much less marked. A tubercle of the mucous membrane of the bronchus may project into its lumen. Owing to the entire occlusion by a submucous tubercle, the bronchus is often found to be surrounded by strips of atelectatic parenchyma. Sometimes an entire lobule is atelectatic. As the result of more or less complete airlessness the bronchi most concerned in the proliferation are collapsed. Atelectasis and marked pathological increase of connective tissue seem to be especially characteristic of the inflammation as it attacks the deep

[1] Karl Ernst Ranke, "Primäreffekt, sekundäre u. tertiäre Stadien der Lungentuberkulose," *Deutsches Arch. f. klin. Med.*, 1916, CXIX, 291 and 297. Also, *Idem*, 1919, CXXIX, 224.

lung in the vicinity of the hilus. Minute calcified foci, sometimes few, sometimes numerous, are scattered through the bands of fibrous tissue in their course between hilus and periphery of the lung. The largest of these foci is generally subpleural, or at least quite superficial.

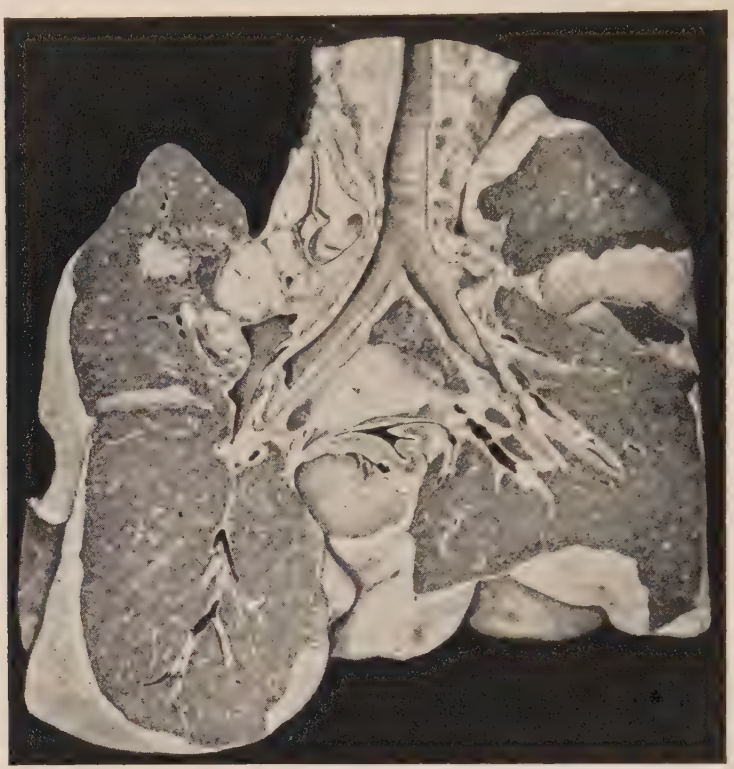


Fig. 25.—Primary caseous focus in the left upper lobe with miliary tubercles in the surrounding lung-tissue. Caseation of the regional bronchopulmonary lymph-nodes on the left upper lobe situated near the opened bronchus that is partly visible. Caseation of the left upper tracheobronchial lymph-nodes and fusion of these lymph-nodes with the mesial surface of the left upper lobe. Acute miliary tubercles in the lower tracheobronchial lymph-nodes. Disseminated acute miliary tubercles in both lungs. The upper tracheobronchial and bronchopulmonary lymph-nodes on the right side are free from disease. The lungs are from a four year old child. (Ghon, "Der Primäre Lungenherd bei der Tuberkulose der Kinder," Urban and Schwarzenberg.)

This focus, usually calcified, is surrounded by abundant fibrous tissue. If the tuberculous process is comparatively recent the tissues are very hyperemic, if old they are dry and fibrous.

Such a process, which may run its course in young children who have never been known to be ill, affords the best opportunity to study an un-

complicated tuberculosis of the resistant individual. For, however minute in size and difficult of detection it may be, it has passed through all of the stages of an arrested tuberculosis of the lung. Such minute tuberculous processes may be found in any part of the superficial lung of the young child and, since they are persistent in the arrested stage, they are also found in the lung of the adult when sought for with sufficient care. Similar processes on a larger scale in the adult show a special predilection for the apices of the lungs. In either case, what we may call the early arrested tuberculous process presents itself as a more or less superficially situated parenchymatous calcified focus, connected by bands of connective tissue with the gland of the hilus to which the affected region is tributary. Pathologically, the lesion is compound; a parenchymatous focus of tuberculous inflammation is connected with a tuberculous adenitis at the hilus by lymph-channels which transmit the infection and, in so doing, are themselves attacked by tuberculous lymphangitis.

Manifestly the findings may be explained in two ways: First, the parenchymatous lesion is primary, the seat of aërial infection with the tubercle bacillus: the gland is secondarily involved. Second, a gland of the hilus which has a tuberculous adenitis of some severity infects the neighboring lymph-channels. The tuberculous process advances as a lymphangitis along certain of these lymph-channels in a centrifugal direction (the current of the lymph being blocked by the adenitis so as to become a stagnant culture medium for the bacillus) until, reaching the periphery of the lung, especially favorable conditions are obtained for the development of a parenchymatous lesion. The arguments for and against these two hypotheses can not be considered here. It may, however, be remarked that primary infection of the parenchyma probably occurs chiefly in very early tuberculosis; infections proceeding peribronchially are most conspicuous in the extensions of an already well-developed tuberculosis. For our present purpose—the consideration of the diagnosis of early arrested tuberculous lesions—the exact manner in which they originate is relatively immaterial.

The distinction between primary and secondary foci in extensive, and especially in fatal, tuberculous lesions is made by the behavior of the lymph glands of the implicated region, which swell greatly and caseate or suppurate in primary tuberculosis and are comparatively little, or not at all, affected in reinfections.

On gross inspection of a small and obsolete apical lesion, a dimpling of the pleural surface of the lung is often seen, with or without a fibrous thickening of the pleura; there is also more or less fibrous thickening of the subpleural tissues. The largest and usually subpleural, or super-

ficially situated, tubercle which identifies the lesion as tuberculous is found sometimes with considerable difficulty and in some cases, which on account of their similarity otherwise to recognized tuberculous lesions are in all probability also tuberculous, it is not found at all. The deeper specks of calcification are generally for the most part overlooked unless serial sectioning or x-ray examination of the block of tissues is resorted to.

Such a lesion, if situated on a surface accessible to physical examination, presents itself as a limited region of increased transmission of sound due to relative airlessness and to increase of fibrous tissue with characteristic prolongation of expiration and harshness of breath-sounds, the changes being only detectable on careful auscultation. Here we have a process of considerable extent in a centripetal direction which is only accessible to auscultatory examination at its superficial end. It appears minute to us because it amounts practically to a long narrow cord examined in cross-section. The auscultatory phenomena, however, show a communication of the periphery with air-tubes larger than bronchioles, and, therefore, more deeply situated than the superficial lesion.

The forms of invasion above described may not be confined to the region supplied by a single or few small bronchi. The lymphangitic extensions may be multiple, in which case the invasion is of a more formidable nature. The prospect of clinical cure without detection is less good in direct proportion to the multiplicity of the avenues of invasion. However, it is not very unusual to find a complicated and rather extensive tuberculous process which has become arrested and has escaped detection for many years.

The lesion which is first discovered after it has become arrested is, of course, a dry lesion. The more or less harshly resonant character of the breath-sounds is such as is to be expected in a dry lesion in which the conduction of sound from the bronchi is abnormally good. If, on the other hand, the lesion is still comparatively recent, it is a wet lesion. In such a lesion fibrous tissue has not reached maturity and the tubes are still more or less coated with moisture. The result of the moistness of the tubes is twofold: first, to hinder the transmission of high-pitched bronchial tones. The weighting of the tube with the liquid lowers its vibration rate just as a weighted string gives a lower tone than one of the same length that is unencumbered. The low-pitched sounds of breathing, the vesicular murmur, are however still transmitted through the air-cells and the breathing, therefore, appears to be normal. Second, the fluid content of the tubes favors the production of moist râles.

If the lesion is truly recent there is of course no question of the conduction of bronchial sound by fibrous tissue; the breath-sounds will be

normal and râles will be the only sign of an active process. The character of the râles is all-important in distinguishing them from adventitious sounds and from the râles of a chronic inflammatory process, "dry" râles.

The râles are rather small, never consonating and break without giving the impression of a distinct delay due to resistance; that is, without a distinct "pop," as if formed in a fluid without marked viscidity. In size, as a rule, they are smaller than dry râles or adventitious sounds. In quality they differ from dry râles in general, including fine crackles, by relative softness of sound. Dry râles give the impression of bursting or of the tearing apart of sticky surfaces; soft râles that of the gentle bubbling of an almost non-viscous fluid. A careful study of the physical properties of râles as manifested on auscultation is absolutely necessary as a preliminary to the diagnosis of early superficial tuberculous processes in the lungs. However, it is comparatively rarely the case that the existence of pulmonary tuberculosis is discovered in the incipient stage of the superficial focus.

Conglomerate Foci.—Tuberculous foci are subject to occasional congestions, the cause of which exacerbations is not well understood. From analogy with the congestion of the tuberculin reaction it may, perhaps, be assumed that they are due to the flooding of the lesion with tuberculous poisons, possibly from the breaking down of some small caseous mass and the escape of its contents into the local lymphatics. Such reactions are often called "allergic" reactions.

The congestion may terminate in resolution. If intense the congestion reveals itself by a seepage of blood into the bronchi which, collecting, is expelled as a pulmonary hemorrhage. Congestion may also terminate in a localized tuberculous pneumonia which may sometimes be detected by the very temporary presence of crepitant and subcrepitant râles. The tuberculous pneumonia may resolve, but more commonly terminates by caseation of the region involved, which in early lesions is usually small. Congestion, pneumonia and caseation involving the tissues most exposed to tuberculous poison because more or less surrounded by active tuberculous foci are the stages of the formation of the conglomerate tubercle.

The congestion of the tuberculous focus, whether it resolves or terminates in caseation, produces wetness of the adjacent air-tubes and favors the formation of moist râles. If there has been a hemorrhage râles will persist for that reason, if for no other, for a week or ten days. These râles are due to the blood effused into the bronchi which, until it is absorbed, acts as an irritant to their mucous membrane.

With reference to the transmission of the breath and voice-sounds the conditions are more complicated. It is important to note that the lesion which we discover for the first time as a manifest caseous focus—whether at the time it is undergoing an exacerbation, or, as a conglomerate tubercle or group of conglomerate tubercles, is subject to more chronic irritation and congestion from the presence of tuberculous poisons—is not an incipient lesion. The tuberculous pneumonia was not a primary, but a secondary process and the caseous mass that results therefrom is surrounded by living tissues, the pathological changes in which existed in part before the advent of the pneumonia. In such a lesion the former discrete foci, now fused into one or several larger caseous masses, were surrounded before their fusion by more or less abundant and mature fibrous tissue. Much of this tissue is involved in the caseation but that portion which borders externally the margins of the now necrosed area still lives and serves as the beginning of the encapsulation which is ultimately to surround the conglomerate tubercle. It may, therefore, be expected that physical examination will reveal the presence of fibrous tissue, the more clearly, of course, the longer the previous duration of the discrete foci, or of the conglomerate focus, and the less the congestion of the tissues concerned. The physical signs about such a lesion while it is comparatively recent will usually be similar to those of the wet fibrous lesion: râles of a more or less "viscid" quality; somewhat more of bronchial quality in the breath-sounds; increased vocal resonance; possibly increased vocal fremitus; a somewhat higher-pitched and shortened percussion-note. However, in the acuter types, or immediately after a marked exacerbation, the congestion may be such that the breathing will appear for the time to be normal and the râles more like those of the incipient lesion. Or changes in percussion-note and in breath-sounds may be noted and ascribed to inflammatory infiltration. If the distinction is not made at the first examination it will become apparent as the congestion of the exacerbation abates, for then, instead of remaining normal or becoming normal, the breathing will be found to have more or less of the characteristics of the fibrous lesion around the dry air-tube, according to the age of the process and the degree of development of the fibrous tissue. In contrast with the incipient lesion there may, therefore, be expected a certain incongruity in the signs and symptoms of the conglomerate focus, which discloses a more complicated pathological history. On the one hand, malaise, fever, hemoptysis, cough with sputum positive for the tubercle bacillus, râles of a type characteristic of the active tuberculous process, speak during the course of the exacerbation for an acutely active process; on the other hand, the type of breathing, and the percussion-note

usually during and always after the exacerbation indicate a chronic element in the lesion.

When the exacerbation has passed the temperature may become normal, the patient may gain weight and feel well, cough may disappear and with it the bacilli-laden sputum. It may appear that the lesion is arrested—a conclusion that it is dangerous to draw. The possibilities of the case have been revealed in one exacerbation; when another may develop it is

FIG. 26.—Large cavities above. Below peribronchial extensions with mature tubercles of characteristic shape. The relation of the groups of tubercles to the bronchi is apparent from their arrangement.

impossible to foretell. The diagnosis "arrested" is, therefore, only properly made after a period of observation sufficient in length to make it improbable that the exacerbation will recur, and also to exclude the probability of extensions having occurred during the exacerbation from disseminations of tubercle bacilli into previously healthy tissues.

After the lesion has become quiet and dry the examiner has the best

opportunity to determine the real extent of the tuberculous involvement by auscultation of the breath-sounds. The extent of the region about the lesion over which bronchovesicular breathing is present should be most carefully determined and recorded for future reference.

Peribronchial Tuberculosis.—The term "peribronchial" may mean either tuberculosis of the coats of the bronchus or of the tissues that surround the bronchus but anatomically do not belong to it. The tuberculosis of the incipient manifest tuberculous process, as already described, is a tuberculosis of the lymph-vessels which follows their course in a general way but makes excursions, when a favorable opportunity arises, into the neighboring tissues. And the inflammatory process seems nowhere to be limited by the walls of the lymph-channels; the proliferation of connective tissue excited by the tuberculous poisons results finally in the formation of a trabecula of fibrous tissue, relatively thick as compared with the diameter of the vessel which primarily conveyed the infectious material. Whatever the views held as to the genesis of the small and early parenchymatous lesion, there can be no doubt that an extension of the tuberculous process may take place peribronchially from the hilus glands on a much greater scale. This process, except for its magnitude, is essentially the same as that which has already been described. It is still a tuberculosis which follows the course of the lymph-vessels but overflows more or less into the surrounding tissues, according to the abundance of the infectious fluids and the degree of resistance of the patient's organism. We may have, therefore, a peribronchial tuberculosis which is practically a tuberculous lymphangitis and which terminates as a broad band of fibrous tissue; or, at the other extreme, one that invades the bronchus to a serious extent, becoming a bronchial, as well as a peribronchial tuberculosis, escapes into the parenchyma and results in extensive pneumonic lesions; that is, in a caseous bronchopneumonia.

In favorable cases the x-ray discloses heavy and broad bands of opacity which radiate out into the lung along the vessels and bronchi, hiding them from sight. It is not unusual to see processes of this kind which involve a considerable portion of both lungs and extend far down into the lower lobes. To the uninitiated the condition of the lungs, as shown by the roentgenograph, seems most serious, yet such a lesion may remain arrested indefinitely and interfere but little with the activities of the patient. Since the fibrous lesion is for the most part deep-seated, the physical signs are few—a slightly bronchovesicular breathing which if closely studied is found to delimit accurately the extent to which the process has approached the surface of the lung, a slight increase in vocal resonance and possibly a perceptible diminution in the expansibility of the lung. If such a peri-

bronchial extension is seen at an earlier stage of its development its activity is shown by râles of a more or less indeterminate character which seem, and are, distant from the ear. A process of this kind is practically as benignant as the minute extension which has already been described, being pretty well confined to the lymphatics. In cases with a greater amount of infectious material or a lesser resistance, the infection involves the parenchyma neighboring upon the affected bronchi and surrounds the tubes with pneumonic foci. We have then a bronchopneumonia which proceeds directly from large bronchi to parenchyma, as distinguished from the more common type of bronchopneumonia in which the inflammation reaches the air-cells by way of the smaller bronchi and of the bronchioles. The former type may be compared with the pneumonic inflammation which surrounds and proceeds directly from the larger bronchi in severe bronchopneumonia following influenza, as contrasted with the more ordinary type of non-tuberculous pneumonia. In peribronchial tuberculosis the pneumonic involvement may be of slight extent. In the roentgenograph the affected bronchi then resemble branches of a tree which are coated with a mossy growth. In other cases larger pneumonic involvements may lead to a considerable widening of the bands of opacity with obliteration of the outlines of the air-tubes. In still other cases the invasion of the parenchyma may be more extensive and irregular and the lung may show at autopsy a series of bronchopneumonic foci of the more usual type which from their position and interrelations are plainly of peribronchial origin. The peribronchial process is the more serious because the deep lung may be equally affected with the superficial lung, the deeper foci being of course inaccessible to physical examination. In the acutest forms of peribronchial extension the disease pursues a rapidly fatal course. The peribronchial becomes bronchial tuberculosis, the coats of the bronchi are extensively infiltrated and stiffened and an incessant irritative cough develops which speedily wears the patient out. The smaller bronchi, now also stiffened by the inflammation, conduct bronchial sounds more perfectly to the surface of the lung. The respiration is harshly bronchovesicular, voice conduction is increased and moist râles are abundant. Here, somewhat exceptionally in tuberculosis, increased conduction of sound takes place through infiltrated rather than indurated tissues.

Peribronchial tuberculosis may develop in a lobe already the seat of parenchymatous lesions. It not infrequently complicates tuberculosis of the upper lobe by incursions into the lower lobe of the same side. For this reason frequent examinations of the supposedly healthy lower lobes should not be neglected. As has already been remarked, peribronchial

tuberculosis is often bilateral. In this case it may be superadded to bilateral superficial lesions and sometimes without adding materially to their gravity. In fact, the curative, but crippling fibrosis of the lung is genetically largely the last stage of an extensive peribronchial tuberculosis. The presence of broad streaks of opacity in the x-ray negative is, there-

FIG. 27.—Peribronchial tuberculosis. Upper lobe a mass of very dense fibrous tissue interspersed with cavities. On close inspection there may be seen relatively recent miliary tubercules in the fibrous tissue which have their origin in the lymph-vessels and are probably due to the dissemination of tubercle bacilli which is characteristic of the complete loss of immunity shortly before death. Below, peribronchial extensions with acinous foci more mature than those shown in FIG. 30.

fore, by no means necessarily an unfavorable sign. The most dangerous, because most easily overlooked, form of peribronchial tuberculosis is the contralateral form. In this class of cases the existing tuberculous lesion may be considered to be unilateral, and the attention of the examiner may be focussed upon that side of the chest until he discovers, to his dismay, that almost the entire lung of the other side gives evidence in the form of râles of a new involvement. It is very common for peribronchial

tuberculosis to develop thus insidiously on the side opposite to an old lesion. For this reason careful examination of the supposedly healthy lung should by no means be neglected. The prognosis of this new extension is not necessarily bad, but if it is of a kind to permit hopes of

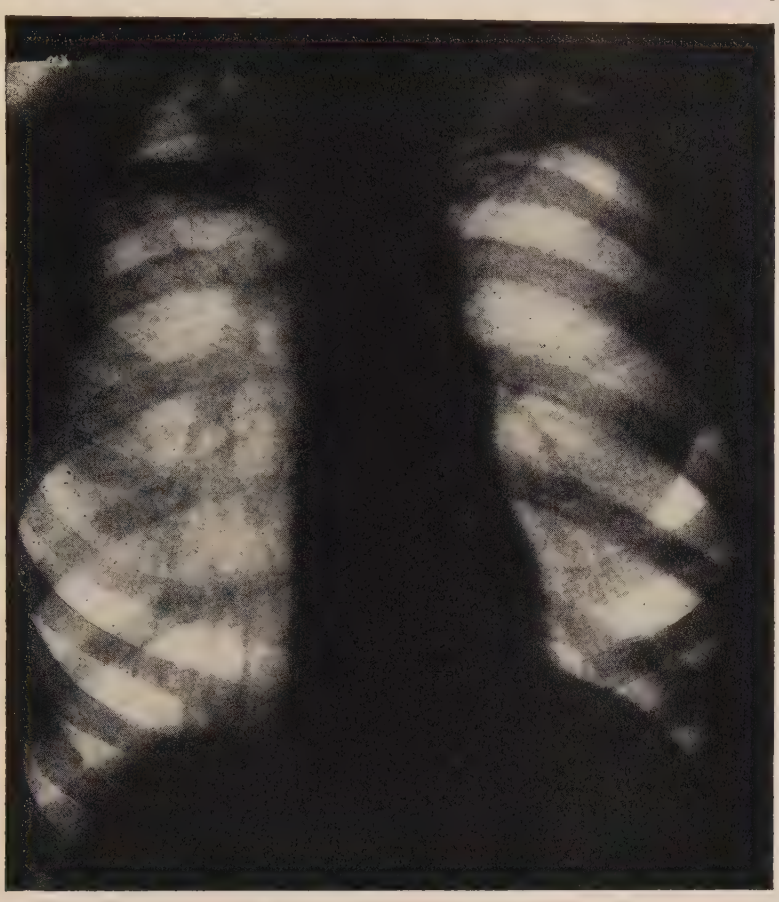


FIG. 28.—Tuberculous peribronchial lymphangitis both lungs, involving especially the paravertebral portions, but also extending to a lesser degree laterally from the hilus with parenchymatous lesions both apices. A few tuberculous lymph-nodes are seen the connection of which with the bifurcations of the tubes may be made out in some instances, especially in the right upper lobe.

recovery the outlook is, of course, good in direct proportion to the earliness of discovery and efficiency of treatment. Sometimes a peribronchial tuberculosis, fatal from the outset, appears in a clear sky when the patient supposes himself to be in good health, but always probably on the basis of an existing tuberculosis of the hilus glands of clinical importance, though exceedingly apt to be overlooked. Since so much of this ex-

tensive process runs its course in the deep lung, the indication by physical signs of its presence are few. There is, therefore, all the more need of painstaking examination of supposedly healthy parts of the lung. The presence of more or less indistinct râles over extensive regions of such a lung is a phenomenon to which most serious attention should be paid. It may mean a bronchitis, but is far more likely to be a sign of tuberculous peribronchitis. The x-ray is an immense help in the diagnosis of peribronchial tuberculosis, as it is in all deep-seated forms of tuberculosis.

The Activity of Tuberculous Foci.—In studying the manifest tuberculous lesion we first ask ourselves, is it active or inactive? By activity is meant the existence of a tuberculous inflammation. A lesion that increases in size by increments of tissue, newly inflamed as the result of the tuberculous infection, is said to be an advancing lesion. In a progressive lesion, activity continues and the disease pursues its regular course of development. It may progress from bad to worse without advancing—progressing, that is, from few to many foci in the same region—but usually a progressive is also an advancing lesion. A stationary lesion is one that does not advance; that is to say, attacks formerly healthy tissue. It may be active or inactive; an inactive lesion is naturally always a stationary lesion.

Activity of a lesion is determined positively by the presence of small or medium-sized râles. The finding in itself, however, simply means that certain patent air-tubes are so irritated by the proximity of some source of irritation that there is an effusion of mucus within them. A sputum positive for the tubercle bacillus is a corroborative sign, but does not indicate the location of the active lesion. The same is true of the focal tuberculin reaction. Such a reaction indicates the presence of tuberculous infection and of a tuberculous lesion, but it does not necessarily indicate that the focus which responded to the injection of tuberculin was at the time the subject of an active tuberculous inflammation. But râles, a positive sputum and a focal reaction to tuberculin (which should very rarely be used in practical diagnosis) together make it very probable that the focus in question is an active tuberculous focus. Activity is determined negatively by signs that indicate that the focus is very fibrous and very dry. If râles are present in the lesion but are coarse and of the character of those produced by a very viscid or tough mucus, it is probable that there is no local tuberculous activity but rather a bronchial irritation due to past damage to the bronchi.

Except for two relatively rare conditions—the dry congestion of an acutely inflamed mucous membrane and the disappearance of râles from the lungs of a moribund patient—a lesion without râles is an inactive

lesion. A lesion without râles is generally a more or less fibrous lesion. We judge of the amount and character of the fibrosis by the characteristics of the breath-sounds and the percussion-note. If all signs end at the same level, which is distinctly marked, the presumption is that the lesion is not advancing though there be signs of activity above that line. This finding does not, of course, exclude absolutely the possibility that recent tuberculous extensions, as yet unmarked by râles, have occurred beyond the boundary line. But that possibility exists with regard to all of the diagnosis in the field of tuberculosis and is to be ignored. The diagnostician is only responsible for the accuracy of conditions which are revealed by indications of some kind and can not guarantee the absolute infallibility of the conclusions that he reaches. In advancing lesions the focus extends downward at least as far as the râles are heard, though dullness and changes in breath-sounds may disappear at a higher level. Such râles will be rather small moist râles, showing a recent more or less acute extension.

It is important that the student bear always in mind the relation of the character of the breath-sounds to the age of the lesion and that the transitions in physical signs which indicate differences of age in the same lesion be noted.

The necessity of noting the age of extensive lesions instead of judging a lesion solely by its extent is illustrated in the following case. A physician once came under the care of the writer, with the prognosis of death within a few weeks. He was thin and weak, had a harassing cough with mucopurulent expectoration, and his chest presented evidence of an extensive tuberculous process. But the lesions were very fibrous and appeared to be stationary; such râles as were present were all coarse and his temperature was normal or but slightly elevated. The case was plainly one in which there was massive fibrosis and in which the cough and expectoration were caused by damaged bronchi, not by breaking-down tubercle. The patient was told that, while he was not likely to recover entirely, no reason was apparent why he should not expect a considerable prolongation of life. As a matter of fact, he lived about ten years, practising his profession almost to the last.

An important aid to prognosis is often neglected by failure to note the presence of pleural adhesions and the degree in which they impair the expansibility of the lung. It is difficult to determine this point by percussion when a parenchymatous lesion lies beneath the pleura, but, in general, firm adhesions give rise to more marked dullness on percussion, since they impair the vibratility of the chest and, unless very thick, they conduct the voice-sounds with greater force, since by the union of the pleural

leaves one surface for reflecting sound has been eliminated. The best test is the functional test. How well can the patient inflate the affected lung when he does his best, and when this is done how do the breath-

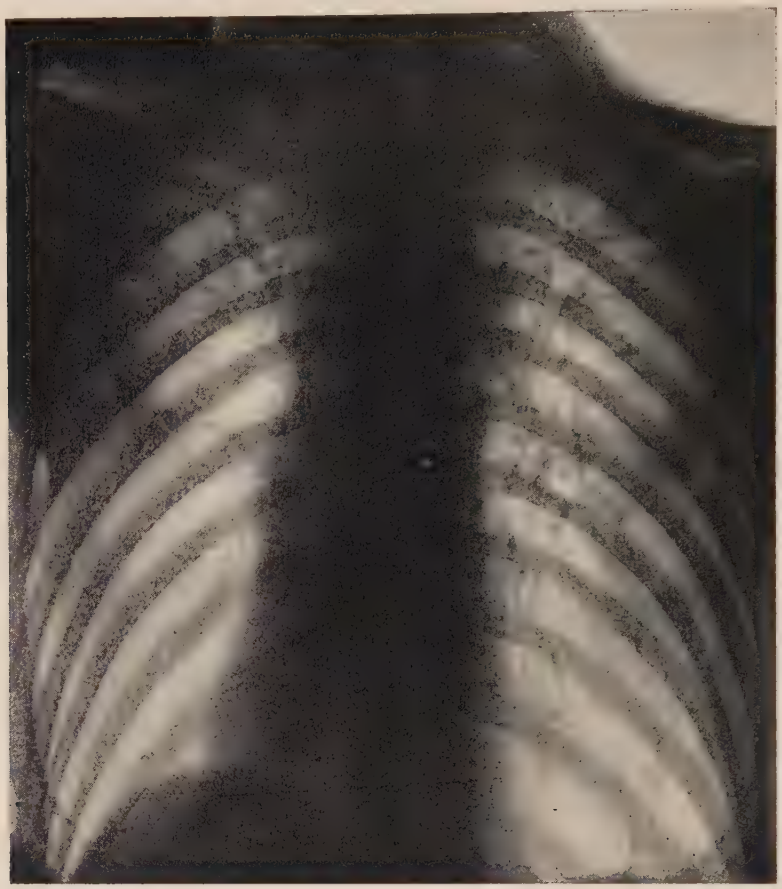


FIG. 29.—Old tuberculous peribronchial lymphangitis involving the upper lobes of both lungs and the paravertebral portions of both lower lobes and terminating on the right, and probably also on the left in lesions of the superficial parenchyma. In the right lung which has the severer lesion the fibrotic areas of the upper lobe nearly coalesce, but the individual streaks may still be distinguished. Healed parenchymatous lesions of the right apex are not well shown. Along the streaks are seen darker spots which represent tuberculous lymph-nodes and which in some places can be identified as such by their situation at the bifurcations of the tubes. Note that the streaks in the lower lobes become fainter and lighter as they progress downwards and for the most part disappear while still far from the diaphragmatic surface of the lung. This case has been entirely arrested for probably at least twenty years.

sounds compare with those of the other lung? Sometimes large and old, rather fibrous lesions are overlooked because percussion has not been

accurate and because adhesions have so tied down the lung that it breathes little except when forced to do so. Breath-sounds may be almost inaudible in such a case in ordinary breathing, because the respiration is feeble and on account of their practical inaudibility they may be judged to be normal. The breathing is also too weak to stir up the perhaps somewhat tenacious mucus so as to produce râles. But, after expiration and cough numerous râles are heard which put quite a different face upon the condition.

The Advancing Tuberculous Lesion.—The manner in which a tuberculous lesion is first laid down often gives a good idea of the degree of resistance of the individual at the time and also of the probable course of future extensions. In case of high resistance the lesions tend to remain localized and will be likely to confine themselves to the lobe in which they originate. Hence, for example, the diffuse fibrosis originally due to lymphatic extensions may remain confined to the upper lobe. When there has been a good reaction from a temporary lapse of immunity we may occasionally find that the breaking down of conglomerate tubercles has resulted in the formation of a cavity which is confined to the upper lobe. But cavities generally progress without regard for the boundaries of lobes and, in general, the more concentrated the tuberculous poisoning, the less the relation that the resulting caseations bear to anatomical dividing lines. This fact is well illustrated by the ease with which a tuberculous focus of the posterior surface of the upper lobe extends across the interlobar fissure to the summit of the lower lobe, as soon as the walls of the fissure have been glued together by plastic adhesions.

Tuberculous lesions extend in two ways: first by continuity; second, peribronchially. The latter mode of extension has already been considered. On the large scale, it is especially frequent in the later stages of tuberculosis and may be regarded in the light of a pathological accident which may or may not especially affect the parts immediately contiguous to an existing lesion. Usually, in the more resistant cases, extension occurs by continuity. The localized tuberculous lesion, if large, may have involved the deep as well as the superficial lung. But if the lesion is small it remains preponderatingly superficial; that is, the largest and sometimes the only lesion lies close beneath the pleura. That this is true is shown by the fact that small dry and old cavities, which may remain as the chief evidence of an early tuberculosis, are usually superficially situated. In the case of a man who had been murdered while apparently in robust health, the only tuberculous lesion of the lung which remained active after a moderately extensive and severe tuberculosis of the upper lobe was a small superficial cavity, the floor of which contained caseated tubercles. In less advanced processes the superficial lung like-

wise bears the brunt of the tuberculous inflammation. It does this because the suction of the thorax in inspiration tends to draw the lymph toward the surface of the lung, this movement being favored by the valves which in the superficial portion of the lymph-tubes open toward the pleura. Early implications of the visceral pleura in the inflammatory process leads to the formation of localized adhesions which limit the expansion of the affected portion of the lung, already more or less stiffened by the inflammation. The diseased region therefore becomes relatively non-expansile and its lymph, which has been increased above the normal by the inflammatory congestion, tends to collect on account of the lessened respiratory movement in the diseased tissues. This region borders upon healthy lung, more actively expansile and relatively poor in lymph. The conditions manifestly favor the suction of lymph from wet to relatively dry tissues and from parts of less to parts of greater motion; that is, from diseased foci to healthy lung. The amount of lymph thus aspirated into normal tissue will depend chiefly upon the force of the respiratory movements of the latter and the abundance of the lymph in the tuberculous focus.

The foregoing explanation is the current one and seems to account best for the superficial location of the early tuberculous foci in extensions of the disease. Concentrated tuberculous poisons, as we have seen, are no respecters of anatomical boundaries. But extensions are usually laid down primarily as discrete and small tubercles. W. S. Miller, an eminent authority on the anatomy of the lung, examining tubercles so small and recent as to permit a study of the progression of the infection—that is, tubercles of miliary size—in every instance was able to trace their origin to one of the situations in which lymphoid tissue is found in the normal lung, namely, at the bifurcations of bronchi, arteries and veins. The infection, therefore, really progresses primarily by way of anatomically recognizable lymph-vessels rather than by direct continuity through lymph-spaces; so that we find that extensions from existing foci take the same course as that which we have followed in the primary invasion, the infection of the parenchyma still resulting from overflows from the lymph-vessels. But from the superficial location of the tuberculous foci it would seem probable that the new infection of the lymphatics which Miller finds does not proceed from the hilus, as in typical peribronchial tuberculosis, but is effected locally. And though infection proceeds from lymphoid tissue situated upstream in the (superficial) lymph-current, laterally-proceeding soakage of the newly invaded tissues with poisoned lymph from adjacent tuberculous foci would naturally diminish the resistance of healthy cells to the infection and favor the processes of casea-

tion. In the resistant individual who avoids strong respiratory movements, extension of the tuberculous focus, if it occurs at all, takes place in continuity; that is, colonies of tubercle bacilli establish themselves as discrete foci in the healthy parenchyma in the immediate vicinity of the diseased tissues. With more active respiratory movements, and especially when there is a lesser degree of resistance on the part of the organism of the patient, new tuberculous foci are more widely scattered. In parts of slight motion and of good resistance the result of the invasion will be relatively few and relatively large conglomerate tubercles; in parts of greater motion the tubercles will be small and numerous. In general, as a tuberculous focus extends downward the individual tubercles become smaller and more closely aggregated; for the increasing respiratory movements oppose the stagnation of the lymph and the individual tubercles are better drained, so that their conglomeration into larger caseous masses no longer takes place. The density of aggregation of the tubercles is the expression of the loss of resistance which permits tuberculous foci to become established in the absence of large accumulations of poisoned lymph and in parts which move with considerable freedom in respiration.

The Mechanics of the Extension of Tuberculous Foci.—Observation teaches that the development of tubercle in the lungs is affected by the amount of motion of the part implicated. Viewed as the field of possible extension of tuberculous processes, the lung may be roughly divided from above downward into three regions: the region of little motion with few, large and well-localized foci; a region of moderate motion with small and more numerous discrete foci and a region of great motion in which tuberculous foci develop with difficulty or not at all. Distinctions as to motility apply, of course, with especial force to the more or less sedentary and physically untrained subjects. When the individual is an athlete in training or engaged otherwise in the most active physical exertion, so that the entire lung is constantly called upon to breathe to its capacity, the above distinctions disappear. It is in precisely such cases that pulmonary tuberculosis, when it is able to develop at all, is found to be of a disseminated and relatively acute type. The exception, therefore, goes to prove the rule.

The pulmonary tuberculosis of the civilized adult is characterized in the majority of cases by a localization of the tuberculous process which is the more marked the higher the resistance of the individual. The ability to limit thus the incursions of the tubercle bacillus has rightly been ascribed to the existence of a relative immunity, acquired in successful resistance to previous infection. Or, to put the matter somewhat differently, when the character of the juices of the patient's body does not

favor the growth and multiplication of the tubercle bacillus, tubercles can only form in places where the conditions are especially favorable for them; so that slight differences as to location, motility and the like become of importance in the pathological development. It is a commonplace to say that the tuberculous focus of the lungs first develops by preference, in the relatively immune individual, in the upper part of the upper lobe. Many explanations have been given of this which can not be discussed here. We will concern ourselves at the present time with the effect of the motility of the lung upon the location and character of tubercle. Evidently bacteria might multiply in a rapidly moving stream of blood. The circulating blood, however, tends to disperse bacteria. That isolated tubercle bacilli are harmless to the relatively immune organism is shown by the frequency of bacteriemia not followed by evil results. To produce the profound effect upon the human tissues which results in the formation of tubercle in the relatively immune individual the enemy must attack in force—there must be a collection of tubercle bacilli with their poisons, for the latter are also of influence. Such collections can only be formed through the instrumentality of the lymphatic system: "Early tuberculosis is exquisitely lymphogenous."

As tubercle bacilli proceed along the lymph-channels which lie between hilus gland and superficial focus it would seem that the conditions are not good for the formation of tubercle, since the tubercles found along the lymphatics are not numerous and are very small. Larger tuberculous foci are found in cases of high resistance only at the ends of these lymph-channels. In some cases the development of an opaque streak extending upward toward the apex from the hilus has been detected by the roentgenologist while its terminal end is still deeply situated, and its slow progress has been traced during weeks in successive photographs until it finally reaches the apex of the lung. In the best cases there is but a single line and that line proceeds straight upward through the paravertebral portion of the upper lobe. We may say then, that, when conditions are unfavorable for the tubercle bacillus, advance, if possible at all, must be along such a line. The explanation of this is simple. The more difficult the growth of the tubercle bacilli, the more necessary that they shall be undisturbed by too rapid motion of the lymph. They must, therefore, advance through lung which is least subject to respiratory changes of volume. The part of the lung that moves least in respiration is the paravertebral portion of the upper lobe which is prevented from expansion inward by the spine, is most remote from the pull of the thoracic wall and is situated too high to be greatly influenced by the diaphragm. And the portion of the superficial lung in which this lymph-

channel terminates—the apex—is the part that moves least in respiration. The tubercle bacilli are naturally not conceived of as having the power to direct their course. They are doubtless dispersed through other lymph-channels but in these fail to find favorable conditions for colonization.

The relative stagnation of the lymph in the upper lung, due not only to inefficient respiratory movements but also to the early formation of pleural adhesions, permits the concentration of tuberculous poisons and at the same time favors their localization by fibrous barriers. This part of the lung is, therefore, characterized especially as the seat of large caseations and of large well-encapsulated cavities, the result of such caseations.

The lower part of the upper lobes and the upper part of the lower lobes is the region of moderate motion. Here the typical tuberculosis appears as smaller and more numerous discrete tubercles. The conditions now do not favor to the same extent large collections of tubercle bacilli and of their poisons. Possibly tubercles are only able to form at all at this location because in the progress of the disease the resistance of the patient is gradually lessening. This view is favored by the fact that downward extensions of tubercle from the upper portions of the lung often does not take place until after the lapse of years.

The base of the lung is the region of great motion. Here, typically, no tubercles form until the immunity of the patient has broken down entirely near the end of life. It is not unusual, in fact, to see cases at autopsy in which the base of the lung is entirely free of macroscopic tubercle although the remainder of the lung is filled with aggregations of tuberculous foci. Here the lower lung, which has acted the more strongly because almost solely responsible for the maintenance of respiration, has been in such active motion and expands and contracts so forcibly that such tubercle bacilli as may have been sucked into it are again expelled before they have the opportunity to colonize.

This is true so long as pleural adhesions do not form in advance of the progress of the parenchymatous involvement. Adherence of the pleura converts parts of moderate into parts of slight motion, so that cavities, for example, are not confined to the upper lung but involve lower portions and are correspondingly increased in size. Similarly, parts of great motion, if their motility is checked by adhesions, lose their immunity from superficial tubercle. Occasionally cases are seen in which the entire pleural cavity of one lung is obliterated by thick plastic exudate. In such cases, if the infection is severe, there are created an enormous number of small tubercles which include the bases of the lungs in their dis-

semination. Life then generally terminates before they coalesce in caseation. But if there is a less acute process large tubercles, such as are generally characteristic of the upper lung, are formed even down to

FIG. 30.—Upper lobe converted into a firm fibrous mass in which there are several cavities. Below, the fine stippling of the caseous areas is due to the involvement of the acini (acinous-nodose form), the slowness of coalescence of the caseated structures showing a chronic proliferative type of tuberculosis. Note that the base is nearly free of tubercle and that the extension downward of the dense foci seems to coincide with that of the adherent pleura.

the vicinity of the diaphragm, thus showing that the differences usually observed between tubercle of the upper and tubercle of the lower lung can be founded upon nothing else than upon the relative mobility of the parts.[2]

TOPICAL DIAGNOSIS

The Apex.—The true apex is that part of the lung which projects above the clavicle at the root of the neck. The apex is not developed in

[2] See Bushnell, "Extension of Tuberculosis in the Immune Lung," *Med. Rec., N. Y.*, March 14, 1914, LXXXV.

the young child, nor is it conspicuous in the deep and short chest in adult life. It is accessible to percussion anteriorly between the heads of the sternocleidomastoid muscle. Posteriorly it extends upward to the level of the first dorsal or even of the seventh cervical spine. Percussion anteriorly is best performed by Plesch's method—orthopercussion. A normal note obtained in this manner is good evidence of the absence of indurative processes of any importance in the upper part of the upper lobe. The contraction of even small lesions in this part of the lung drags the apex down from its normal position. In upper lobe lesions it is more frequently the case that the apex thus disappears than that it remains accessible with impaired percussion-note. A tuberculous lesion is very rarely confined to the true apex.

The term "apex" is usually applied loosely to denote that portion of the upper lobe from which sounds of respiration and percussion are reflected upward to the tissues of the supraclavicular fossa. As has been pointed out elsewhere, except for the part known as the true apex, the upper edge of the upper lobe lies behind or below the clavicle. The condition of this part of the upper lobe is, however, conveniently determined by auscultation and percussion over the supraclavicular fossa and there is no objection to this application of the term "apex" if the anatomical facts are borne in mind. The word will be used in the sense of the above definition in the remainder of our discussion of the subject.

In chronic and well-localized pulmonary tuberculosis the apex is usually the seat of the first manifest parenchymatous lesion which may or may not be confined to this part of the lung. If the lesion is active and of considerable extent there is usually no difficulty in determining that it is tuberculous. But the apex is often found to bear evidences of small and healed indurative processes, with regard to the etiology of which there has been much debate. There are two hypotheses: first, that the induration is due to mouth-breathing; second, that it is due to tuberculosis. Krönig has proposed as a nosological entity a disease which he calls "collapse-induration" of the apex. In his view mouth-breathing leads to dryness and irritation of the respiratory passages which also involves the apices of the lungs, the right apex being especially affected on account of the greater diameter of the right main bronchus. This irritation leads to proliferation of connective tissue ending in fibrotic changes. Atelectasis also is caused by the relative disuse of the apices in respiration. According to this hypothesis, then, the morbid changes are due to a combination of the two factors, fibrosis and atelectasis. This view is ardently espoused at the present day by some French writers. The difficulty with it is that it connects apical changes with obstructive lesions

of nose and nasopharynx. Such connection may seem close in the class of patients that frequents nose and throat clinics, but investigation of the general population discovers many individuals who are not and never have been mouth-breathers and are not known to have been ill, but who nevertheless have apical induration.

FIG. 31.—Topography of the apices of the lungs. (Goldscheider, Berl. klin. Wochenschr., XLIV.)

With regard to the second hypothesis; Bard of Geneva called attention many years ago to what he called "abortive" tuberculosis of the apex, an arrested form often found to be confined to that part of the lung. The observations of Bard are no doubt correct; there is such a tuberculous lesion. But are all such indurations to be considered tuberculous? Until recent years the great frequency of apical indurations has led many to deny that they could represent past tuberculous infection. There is no well-established pathological fact that has won its way with greater difficulty than the fact that almost all, if not all, civilized adults have experienced an infection with the tubercle bacillus. The fact being now perforce admitted, it seems to be equally hard to accept the view that, in a large percentage of cases, such infection reveals itself in the form of an obsolete parenchymatous tuberculosis of the lung, as the visible expres-

sion of the relative immunity against farther attacks of the disease which is so valuable an acquisition for the individual.

In a small percentage of cases the x-ray fails to show connection of the old apical lesion with the hilus, but usually in apical induration one or more well-marked lines of opacity, representing a healed tuberculous lymphangitis, unite the superficial lesion with the hilus glands. Such findings in the roentgenograph speak strongly for the diagnosis of old tuberculous infection. It is true that deep-seated thickening of the lines that extend outward from the hilus may occur in other infections besides tuberculosis but they are not so strictly localized. Widespread formations of fibrous tissue, as shown by the x-ray, may be open at times to doubt as to the infectious agent; an x-ray picture that shows a sharply marked focus of the apex connected by sharply marked lines with the root of the lung—the rest of the lung-field being clear—almost always means a past tuberculosis; the absence, however, of such typical markings does not exclude tuberculosis.

The percussion-note of both apices is normally somewhat higher in pitch than that of the lower part of the lung; that of the right apex is often shorter and higher pitched than that of the left apex. Similar changes occur in the breath-sounds of both apices, which have somewhat of a bronchial quality, on the right to a greater degree than on the left. These peculiarities are more marked in long and narrow chests. It is a much debated question whether the difference in physical signs observed in the comparison of the two apices is a normal difference based on anatomical structure, or whether the percussion-note of the right apex is duller and the breath-sounds more bronchovesicular than over the left apex because the former is more frequently diseased. Viewed as a normal phenomenon, the disparity in physical signs of the two apices has received various explanations, of which two will be mentioned. First, the fact has been referred to that the trachea grooves the right upper lobe; it is in less close apposition with the left upper lobe. (See p. 111.) Tracheal sounds are better transmitted on the right; the percussion-note has more of the tracheal quality. Second, the bronchi on the right apex are of larger calibre than those of the left apex; hence the amount of parenchymatous tissue is relatively greater on the left side. There will be better transmission of bronchial tones on the right; more of the vesicular murmur on the left side. Both of these anatomical differences probably influence the physical signs; the relation with the trachea is the more important one.

In the diagnosis of lesions of the apex the examiner first asks himself the question: Are such differences, as may be detected in the physical

signs as compared with the normal lung elsewhere, ascribable to the anatomical structure solely? If this be the case the differences between the two apices will be slight, being such as would be expected when varying thicknesses of a sound-insulating material (in this case the air-cells) are interposed between the ear and the source of sound. In respiration and on percussion both apices will furnish bronchial tones more marked than in normal respiration elsewhere, but as has been remarked, the difference between the right and the left apex will be slight. As the bell of the stethoscope is moved from place to place over the same apex a difference as to loudness and quality of breath-sounds may be detected. Such changes as there may be will always be slight and gradual; there will be no abrupt transitions and the differences between the various points of auscultation, if properly observed and properly plotted, will not result in an

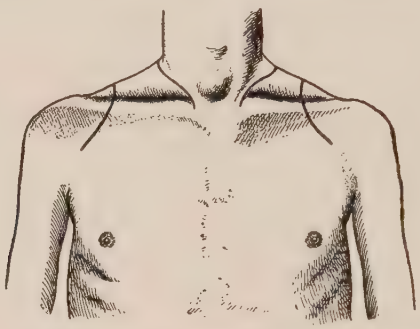


FIG. 32.—Reduction of the left apical isthmus by encroachment of the median and lateral borders in shrinkage of the apex of the left lung. (Krönig, Deutsche Klinik, Berl & Wien, XI.)

irregular figure. Changes in the transmission of the voice are detected with difficulty on account of the proximity to the source of sound. If found to be present, there will be no abrupt transitions between varying degrees of loudness. On the other hand, if the character of the breath-sounds depends upon the presence of indurated tissue, unless both apices should be indurated to a practically equal degree the differences between the two apices will be more distinctly marked both as to percussion-note and breath-sounds and, examining the affected apex topically, it may be found that the transition between the breath-sounds obtainable in the various points of application of the stethoscope are relatively abrupt. It will be possible in this way to mark out roughly the places where the fibrous tissue is densest or approaches most nearly the surface. Similarly, the voice-sounds which find in the indurated tissues more favorable paths of conduction will be transmitted more distinctly—this is especially true of relatively high-pitched tones—over some parts of the apex than over

others and the transitions between the two regions will be relatively abrupt. Percussion may well be used in this examination to discriminate between the notes of the two apices. Topical percussion of the individual apex anteriorly is hardly a practicable procedure, on account of the smallness of the surface available in the supraclavicular fossa and the fact that the percussion-note normally becomes duller when bones are approached.

Evidences of fibrous thickening having been found and it having, therefore, been decided that a lesion exists in one or both apices, it may be inferred under ordinary conditions that it is of tuberculous origin, the theory that mouth-breathing leads to structural changes in the apices being an unproved and improbable assumption. The question then arises as to the activity of the lesion. Since such lesions are found to be exceedingly common in healthy individuals if carefully looked for, the probability is that the lesion is an arrested lesion. However, the opposite assumption, namely, that the physical signs which really indicate fibrosis are signs of an incipient process, is frequently made. More than that, the great importance given to special modes of percussion in the examination of the apices rests upon the assumption that percussion, if only skilfully enough performed, is capable of revealing an incipient tuberculous process in advance of other signs, the process being usually assumed to have been initiated as the result of direct aërial infection of the apex in the immediate past. This is a delusion. Percussion does not discover a new tuberculous infection of the apex in advance of auscultatory signs. This is shown to be true by the well-proven and long admitted fact that percussion is powerless to detect the countless tubercles of miliary tuberculosis in the early stage of the disease and the small tubercles that characterize the early stage of the extensions of an active tuberculous focus. If it cannot do this for what reason is it to be expected to be more successful in the smaller lesions of incipient apical tuberculosis? If bronchovesicular breathing and a shortened and raised percussion-note are present, these are almost always signs of an old, not of a recently developed tuberculous focus. This is practically certain to be true if there are no râles and if the patient is afebrile. The exceptional possibility that these signs may be due to a pneumonic infiltration has already been discussed. Tuberculous pneumonia of the supraclavicular region is excessively rare and when present is here, as elsewhere, not the sign of a new but of a relatively advanced lesion, although in active and acute processes it may appear when the infection has been of short duration. The x-ray is very helpful here if doubt exist. A sharply marked apical focus connected with the hilus by sharply marked lines is always an old and ar-

rested lesion. Apical tuberculosis in its initial development is frequently accompanied by apical pleuritis which results in adhesions. These may produce opacities in the roentgenograph, but so far as opacity is due to the pleura it is persistent and uniform in density. Pleural adhesions restrict the movement of the lung. This restriction may show itself in two ways: first, by diminution of the breath-sounds; the breathing is weak. Second, by interference with the circulation of the blood. Sluggishness of the circulation of the blood leads to increase of the lymph and thus to a general "sogginess" of the part. The congestion may manifest itself by a uniform opacity to the x-ray. When respirations deeper than usual relieve the congestion the opacity, so far as it is due to congestion, disappears. Increase of the fluid contents of the lungs beneath adhesions, in breathers who aërate the apices imperfectly, often leads to the formation of more or less tenacious mucus in a quantity sufficient to produce râles which are most marked during the first deep inspirations. These evanescent râles, when the fluid in the bronchi is small in amount and sticky, may resemble dry crackles. In other cases rather abundant mucus is sometimes found at autopsy in the tubes directly under localized adhesions but not elsewhere in the lungs, there being no active tuberculosis whatever. In such cases, examined during life, numerous moist râles may be a fruitful source of error in diagnosis. The correct diagnosis can not always be made from physical signs at the first examination. It is based on the discrepancy of the signs. The râles, if moist, may be such as would be expected in an active process, the breath-sounds are those of an arrested process; the patient is quite afebrile and his general health may be good; the sputum is scanty and mucous and is generally negative for the tubercle bacillus. Moist râles, though sometimes very well marked in the apex, are more characteristic of lower levels of the lung. The distinction between râles due to congestion and small or medium-sized râles from bronchi irritated by their proximity to breaking-down tubercle is sometimes very difficult. The former cause is the probable one if the patient is in vigorous health and no other signs of activity are present.[3] Prolonged observation may be necessary in order to establish the diagnosis. Fine crackles in the apex, disappearing after a few breaths, are sometimes called "unfolding" râles on the assumption that the inner surfaces of bronchioles or air-cells, which are in contact because of the relative airlessness on deep breathing, yield sounds due to the separation of sticky membranes. These râles are also called "atelectatic" râles. Unless bronchi are actually plugged, however, the lung is not thus readily

[3] In such cases, especially, sounds arising in chondrosternal and other joints should be carefully excluded. See pp. 112 and 113.

deprived of air and true atelectasis is not dissipated by a few breaths.

Study of the sound fields on the top of the shoulder (Krönig's isthmus) [4] throws much light upon the nature of the apical tuberculous lesion. Krönig's isthmus is narrowed in the contraction which results from the chronic tuberculous process, and such narrowing is permanent. In this case the lines of demarcation between dullness and normal resonance are sharp. The acuter forms of tuberculosis are not accompanied by contraction of the isthmus, but a chronic tuberculous process may still be in the active stage yet show narrowing. In this case the contraction is generally less great than when it is a sign of old fibrous changes and the lines of demarcation are "washed," that is, blurred.

From the fact that the apical lesion is usually found in a state of arrest it is often not detected by those who fail to scrutinize with sufficient care rather unobtrusive alterations from the normal of the breath-sounds. Later extensions lower down in the upper lobe may then be taken to be the first of the superficial lesions. For this reason the doctrine is held by some that the earliest evidence of tuberculosis is to be found at the base of the posterior aspect of the upper lobe (level of the third dorsal spine) or even in the upper edge of the lower lobe (posterior surface). This occurs occasionally, but the x-ray shows that in the great majority of cases the summit of the upper lobe—what we call the apex—is the first of superficial parts to be involved.

Apical lesions, outside of the true apex, being situated for the most part below the level of the clavicle, are usually accessible to auscultation and percussion in the subclavicular fossa and, since the edge of the summit of the lobe is relatively thin, they are also accessible posteriorly. Unless the lesion is quite small, evidences of its existence are usually to be found on both the anterior and the posterior surfaces of the chest. The apical lesion often simply represents the oldest portion of a lesion which extends farther down into the upper lobe.

Upper Lobe Lesions.—In discussing the topical diagnosis of upper lobe lesions much of that which has been said as to the diagnosis of apical lesions is applicable. One outstanding difference is, however, at once apparent. When we come to the examination of parts of the lung below the summit of the upper lobe we are no longer obliged to make allowances for normal variations from the standard physical signs of the lung; we expect normal resonance and the normal vesicular murmur over the remainder of the chest with the exception of the slight and localized variations which have been discussed in the chapter on the examination of the healthy chest.

[4] Discussed more fully on p. 116.

Often the tuberculous lesion of the upper lobe has consisted in the deposition of a few tubercles which remain discrete, become calcified, or if small are transformed into fibrous knots. The existence of a lesion is then to be determined solely by the signs that point to diffuse thickening of the connective tissue. In this case we do not identify the individual foci. As the lesion is a dry and old lesion bronchovesicular breathing is distinctly to be made out: both inspiration and expiration are harsher than normal; that is, have a bronchial quality and the expiration is somewhat prolonged. On percussion, comparing the diseased area with the opposite side on the same level or with another normal part of the lung, it is found that the note is shorter in duration and higher in pitch. It is surprising how slight indurations are capable of influencing distinctly the physical signs and how clearly they are shown by the x-ray. Sometimes the measure of the degree of induration and contraction is better afforded by the narrowing or disappearance of Krönig's isthmus than by the physical signs, which may be obscured by the development of compensatory emphysema of the surrounding lung. Such lesions are of no importance to the individual, unless the error be committed of regarding the physical signs as evidence of an incipient or active process. As described, they are never signs of activity. As has been remarked, if they were due to inflammatory infiltration râles would be present and the patient would be feverish and evidently ill.

In estimating prognosis we should consider the intensity of the now obsolete process as carefully as its extent. There is a wide difference between the lesion in which the pleura is glued fast to the ribs, the breathing is harsh, the expiration is much prolonged, dullness is marked and many strongly marked lines appear in the roentgenograph, and one like that referred to above. The second type may be neglected; the first type has some clinical significance for the reason that such a lesion means a crippling of the lung, considerable loss of respiratory surface and, more important, such a severity of the early infection that the persistence of tubercle, however well walled off at the time, is a more or less strong probability.

Bilateral Lesions.—All pulmonary tuberculosis is essentially bilateral, so far at least as the deep lung is concerned. The x-ray shows involvement of both deep paravertebral regions, but more marked, as a rule, upon one side than upon the other. On the side less involved the process may remain restricted to the deep lung, or in time it may in its turn reach the apex and develop superficial lesions of the upper lobe, which do not differ in their pathology from those of the lung first attacked. As a rule, lesions of the lung secondarily involved are larger and more acute

than those of the side first affected. It is a very common error to overlook the initial superficial lesion and to consider the second as the first involvement, or, if the presence of a small lesion is discovered on the side really first implicated, to consider it secondary in point of time to the larger lesion. Unless both lesions are very old the distinction is made as follows: the older lesion will show obliteration or greater contraction of Krönig's isthmus, a harsher breathing and a more prolonged expiration.

Although the first extension from the hilus is peribronchial, after the period of manifest tuberculosis is reached peribronchial extensions are very frequently secondary to superficial and more or less circumscribed lesions.

When a tuberculous process has run its course and become arrested, the process having been sufficiently marked to arouse successfully all the defensive powers of the organism, the result is a certain local immunity. Tuberculosis will not develop anew at this place as readily as elsewhere in the lung. If, therefore, a patient presents evidences of constitutional impairment—fever, lassitude, purulent expectoration, etc.—and examination shows a completely dry lesion of, say, the right upper lobe, it is not permissible to ascribe the symptoms to this lesion, for an arrested lesion has no symptoms. Especial care should be taken to examine the opposite side—in this case supposedly the left—for the slight evidences of peribronchial processes which are so readily overlooked by the careless observer.

In very old and slowly progressive lesions both lungs are usually implicated to a considerable extent. It not rarely happens that such sluggish lesions, whether unilateral or bilateral, give what seems indefinite and scanty information on physical examination so that, while there is no doubt as to the existence of the lesion, the determination of its age, distribution and degree of activity is another matter. If one examines such a lesion dullness is present nearly everywhere over the thorax; breath-sounds are faint or inaudible over the greater part of one or both lungs: râles are of a more or less indefinite character, found here and there, differing in number and distribution in successive examinations; the conduction of the voice is rather diminished than increased.

In such a lesion the difficulties of diagnosis are largely due to the presence of extensive plastic pleural exudates which act in various ways to obscure the physical signs and at the same time often cause the lesion to pursue a very sluggish course. In studying such cases, determine first which lung has the older lesion. Often the patient knows only of the older lesion, believing his disease to be unilateral. Fibrosis and cavity above, succeeded below by well-marked bronchovesicular breathing

throughout the lesion, the lower edge of which is sharply demarcated—the breathing at the base being free and exaggerated, or puerile in character—are favorable signs, for they show that the lesion is probably stationary. The prognosis of this lesion depends chiefly, however, upon the number and size of the râles which follow cough. More often the lesion is a wet lesion, approaching in its character that of the newer lesion of the other side. The more recent lesion is characterized usually by absence of cavities—or cavities when present will be recent—and by feeble respiration with inability to increase its force above a certain slight degree. Often the examiner will believe that the patient is making no effort to breathe until comparison with the other base shows powerful respiratory movements with correspondingly loud breath-sounds there. The relative degrees of dullness on percussion and the presence or absence and distribution of vocal fremitus furnish clues as to the distribution and thickness of the plastic exudate of the pleura. In general the prognosis as to prolongation of life is better the more obscure the physical signs and is to be determined partly by the amount of fibrosis present but chiefly by symptoms (fever, emaciation, loss of strength, etc.) rather than by signs. Here, as in other advanced lesions, the observer should be on his guard against drawing favorable conclusions from the absence of signs of moisture if the patient's general condition is distinctly bad.

In studying these lesions Krönig's isthmus may not indicate the relative age of the two lesions, being contracted on both sides. The older side may show a sharper breathing, a smaller lesion, a free base, perhaps a larger and older cavity. The amount of fibrous tissue should be determined. Extensive areas with dullness and harsh breathing but without râles is a favorable finding. Much less favorable is a wet lung, the bronchovesicular quality being absent or but slightly marked in the respiration. Distinction is to be made between showers of medium-sized râles after expiration and cough, which show inflammatory reaction or softening tubercle, and the few and coarse râles of cavity or large bronchus.[5] The degree of activity of the tuberculous process will probably be best determined by the condition of the lower lobe of the more recently involved side. The effect of extensive pleural adhesions in restraining breathing permits but little information to be obtained from the study of breath-sounds. Percussion gives more useful information. One distinguishes the very dull note of plastic exudate from that of the more resonant parenchymatous lesion. The patient's general condition should

[5] For marginal sounds which should be carefully distinguished from râles of lower lobes see pp. 120 and 167.

be considered carefully in the prognosis and brought into relation with the physical signs.

There is one type of tuberculous lesion somewhat similar to that just described which presents even greater difficulties in diagnosis and prognosis. It may be described as a lesion which has all the evidences of advanced disease: dullness, changes in breath-sounds, abundant moisture, sputum positive for the tubercle bacillus, etc., but in which the patient's

FIG. 33.—Fibrosis and caseation. The lung is practically completely fibrocaseous, the numerous cavities representing, of course, the former site of caseous foci.

general health is good entirely out of proportion to the apparent gravity of the lung conditions. The writer has in mind one patient in whom the breathing of the right lung was greatly restricted and the lung was everywhere full of râles, yet there was rarely fever, the nutrition and appetite were excellent and the patient, except for shortness of breath, was little conscious of ill health. Here we have a case in which firm adhesions interfered with the play of the lungs and led to a stagnation of the lymph, the moisture being apparently due chiefly to such stagnation rather than

to the exudation of active inflammation. The rationale, in short, is the same on a larger scale as in the case of râles produced beneath the adhesions of an arrested lesion. The determination of such cases is made by the dissociation of signs and symptoms. The patient's condition from the physical signs appears to be desperate, but the symptoms indicate a very tolerable, though of course precarious state of health which may be enjoyed for some years. Here as in all advanced cases, symptoms are the surest guide to the prognosis.

Fibrosis and Caseation.—The formation of fibrous tissue in the presence of tubercle is evidence of the power of the patient's body to react to the poisons of the disease by proliferation. Fibrosis is, therefore, in a sense a measure of the resistance of the individual. Caseation, on the other hand, marks the death of body cells which have been fatally poisoned. It is, therefore, a measure of the success of the tubercle bacillus in the more or less distant past in overcoming the resistance of the patient.

Fibrous tissue in general is characterized by relative non-vascularity, contractility and density, as compared with normal pulmonary tissue. On account of these properties the fibrous lesion is relatively dry and because its air-content is diminished or absent, it conducts the sounds of breathing and the voice much better than normal lung. The characteristics of fibrous lesions are: a relative scarcity or absence of râles, dullness on percussion and—since more of the bronchial element and less of the vesicular element of the breath-sounds is transmitted—a higher-pitched or sharpened inspiration and a prolonged as well as sharpened expiration.

When the tissues of the region of the lung examined consist of fibrous tissue which has extensively replaced the air-cells the condition is known as fibrosis. The same term is applied to conditions of less severity, so far as the past pathology is concerned, in which the existence of a former lesion is only or chiefly to be made out from the signs arising from the presence of a disseminated fibrous tissue, the air-cells for the most part still functionating.

Fibrosis may be disseminated or massive, recent or mature. In disseminated fibrosis fibrous tissue has formed about discrete tuberculous lesions, in the adventitia of blood-vessels and bronchi and also results from a thickening to some extent of the connective tissue sheath of lobes and lobules. If the lesions are small and deeply situated no indication of the presence of fibrous tissue may be given by physical signs. Tuberculous processes, however, tend to advance to the periphery of the lung. Even, therefore, in healed lesions we may expect that fibrous strands will extend to the surface of the affected lobe. The effect of any considerable

increase of fibrous tissue in a lung lobe will be an impairment of its vibratility as a whole and consequently a diminution in the transmission of the fundamental note of the thorax, as well as an increased conduction of sounds from the bronchi. A disseminated fibrous lesion will, therefore, be characterized by a shorter and higher-pitched percussion-note than that of the corresponding healthy lobe on the other side of the body and by breath-sounds which have more of a bronchial quality. Expiration alone, or both expiration and inspiration, will show this change; expiration will also be abnormally prolonged. In disseminated fibrosis the above changes will be slight and may only be detected by close observation and careful comparison with the other side. Uncomplicated fibrosis is not accompanied by râles. The lesion, as it presents itself, is an obsolete lesion.

Massive fibrosis is in general of two kinds: first, a fibrosis which permeates and surrounds parenchymatous pulmonary foci. Second, a fibrosis which extends along the bronchi and blood-vessels. In the first form the fibrosis represents the reaction of the patient to a high degree of stimulation; that is, stimulation from the abundant poisons of large foci. The encapsulation-zone forms at the optimal distance from the focus—near enough for high stimulation, not so near that that newly forming tissue can not survive before a too concentrated poison. The larger the caseations the less likely a successful resistance as evinced by ability to proliferate connective tissue freely—the tuberculous foci must not be too large and, especially, they must not be too closely grouped. In the second form the fibrosis about air-tubes and blood-vessels shows itself as massive trabeculae which appear in the roentgenograph as broad dense streaks. This form is due to the stimulation of tuberculous lymphangitis and of numerous small peribronchial tuberculous foci, the result of an overflow to the parenchyma of the tuberculosis of the lymphatics. The smallness of the discrete foci permits them to be easily surrounded by and engulfed in the developing fibrous trabeculae, so that they ultimately become calcified or fibrous. Strictly speaking, in the fibrosis is also comprised lung carnified from atelectasis and organizing pneumonia. The massiveness of the fibrosis is not due solely to the proliferation of connective tissue as a procedure of defense, but in part it represents the remains of airless parenchyma, collapsed bronchi and the like. All the elements, however, are fused into a practically homogeneous mass from the physical standpoint, so that clinically the process may be regarded as a pure fibrosis, which, being deeply situated, because surrounding the larger tubes and vessels, furnishes so few signs of its existence that its extent and density, as revealed by the x-ray, often surprises the diag-

nostician. Clinically, when we speak of massive fibrosis we mean usually the fibrosis which surrounds or replaces parenchymatous foci and extends to the surface of the lung.

The physical signs mentioned as characteristic of fibrosis are those that accompany mature fibrous tissue. Recent fibrous tissue is hyperemic and therefore wet. Small moist râles are therefore present. In view of the slowness of formation of fibrous tissue, it does not often happen that recent fibrous tissue is differentiated as such by physical diagnosis, the signs being in general those of a more or less active and acute inflammatory process surrounding a tuberculous focus.

The effect of the presence of caseous foci is to produce a wet lesion in the case that the cheesy matter is breaking down or that its poisons irritate the neighboring bronchi. A caseation may, therefore, give rise to numerous moist râles and be characterized by an abundant mucopurulent or frankly purulent expectoration. If, however, the caseous tubercles are surrounded by an efficient capsule of fibrous tissue they may give no indications of their presence. Where fibrous tissue predominates the lesion is said to be fibrocaseous. When the caseation governs the physical signs the lesion is said to be caseofibrous. However, in both fibrocaseous and caseofibrous lesions the fibrous tissue generally predominates as to actual mass. The practical distinction between the two classes is really that in fibrocaseous, or dry, lesions the encapsulation shuts off the caseous masses from their environment, while in caseofibrous, or wet, lesions the encapsulation has been penetrated and some or all of the caseous masses are softening and poisoning their surroundings. The larger the amount of caseous substance the less efficient is its encapsulation and the more likely is it to break down.

The presence or absence of râles furnishes a rough guide as to the amount of caseous tissue present in the fibrosis and particularly as to the success with which it is being shut off by the encapsulating fibrous tissue. The prognosis of a wet lesion is much worse than that of a dry lesion. Not only their presence but the character, size, distribution and constancy of the râles must be carefully noted in estimating their significance. Relatively large, viscid, widely distributed and inconstant râles with sputum raised with some difficulty and rather mucous than mucopurulent denote usually a chronic bronchitis of relatively slight importance. Large viscid râles (but less viscid than those just mentioned) constantly present at certain locations with a more abundant, more easily raised and more purulent sputum probably indicate cavity, bronchiectasis or other permanent local damage to bronchi. Small to medium-sized moist râles occurring alone or mingled with larger râles, constant as to location and

more or less constant as to their presence from day to day, denote localized irritation of the smaller bronchi, due in all probability—in a fibrous lesion—to the presence of softening tubercle or of small cavities the result of such softening. Such a finding is more likely, however, to indicate a spot of tuberculous inflammation of the parenchyma. The differentiation is made from the character of the tissue surrounding the region in question, as determined by physical signs.

The elements of the tissues which govern the physical signs of fibrosis are no longer taking an active part in the morbid process. In so far as

FIG. 34.—Caseous pneumonia. Complete caseation of the upper lobe; terminal stage of an acute tuberculous pneumonia. The lower lobe is much contracted from hardening in alcohol, the upper lobe hardly at all.

the condition of the lungs depends upon the fibrous tissue, the lesion is a cured lesion.

It is, however, true that dryness of the tissues, as shown by absence or "dryness" of râles, may be simulated by an intense congestion which as

yet has not relieved itself by an effusion of mucus. A familiar illustration is the exacerbation from chilling of the surface of the chest of a region of the trachea damaged by an antecedent tracheitis. Here we have at first an irritative and unproductive cough, followed later by the expectoration of a tenacious mucus which gradually becomes more fluid and mucopurulent. Dryness from congestion is a part either of an acute process or of an acute exacerbation of a chronic process. The history and symptoms of the patient should prevent any confusion in the differentiation of the dryness from congestion from the dryness of fibrosis.

Caseous Pneumonia.—Caseation in general has been considered in connection with fibrosis, but brief reference should be made to the diagnosis of a purely caseous lesion. The caseous pneumonias, which are the cause of the conglomeration of tubercle and of the fusion of groups of conglomerated tubercles, are only to be identified when on a small scale by following the course of the pneumonia before it has reached the stage of caseation. Large caseous masses, however, present a clinical picture which requires independent identification. It is not very unusual for an entire lobe to be converted into a single caseous mass as one of the acuter manifestations of tuberculosis. In such a case the complete caseation is the last stage of a complete infiltration. But in another type, which is not so common nor so well recognized, autopsy may find caseation in progress but not yet complete. In some regions the tissues are already converted into cheese, other regions have a frosty look showing that the process of caseation has begun but is as yet far from complete.

A caseous mass is absolutely inexpansile. A completely caseated lobe will, therefore, give no autogenous breath-sounds whatever and the breath-sounds of the incomplete caseation will be exceedingly faint or non-existent, according to the amount of the tissues which has not yet become caseated. Cheesy tissue, however, will conduct sound; bronchial breathing and bronchial râles may be heard through it. Dullness, inexpansibility, absence of breath-sounds, or distant bronchial breathing with or without coarse râles, constitute the group of signs that is to be expected. The voice does not appear to be well conducted through massive caseation, but the condition of the patient is usually such that this sign can not be sought for, or at least is not satisfactorily developed. The disappearance of breath-sounds from regions where they were formerly heard and the loss of expansibility of a part which formerly expanded in respiration are the most significant signs of massive caseation.

Cavity.—A cavity represents the terminal stage of caseation, the caseous matter finally being absorbed, or less frequently being expectorated. Cavities may be of acute or chronic formation, large or small, superficial

or deep. The type of cavity which is generally meant when the term is used, and which alone is of any considerable clinical importance, is the large chronic superficial cavity. In this type the superficial lung is destroyed and the outer wall is formed by little more than the visceral pleura which is firmly adherent to the costal pleura and to the underlying ribs. The outer wall of the cavity in this case may be described as firm and elastic. The remainder of the walls of the cavity is made up of fibrous tissue which represents the surviving portion of the former encapsulation; a wall the thickness of which depends largely upon the age of the lesion. Sometimes a cavity, the walls of which appear in the roentgenograph to be of extreme thinness and delicacy of construction, is found at autopsy to have rather massive walls. Here the shadows of the x-ray have been due more to the deposition of mineral salts in the lining membrane and in the immediately adjoining older portions of the fibrous wall than to the fibrous tissue per se. It would be nearly correct to say that a thin-walled cavity is a comparatively recent cavity and that a cavity which appears to the x-ray to be thin walled is more recent than one the walls of which appear in the same illumination to have a more considerable thickness. It is sometimes loosely said that a cavity is a sign of a relative immunity to tuberculosis. It is not, however, the death and disappearance of the tissues that formerly occupied the space which shows an augmented resistance, but the strong fibrous wall of demarcation about the dead tissues. This proves the immunization has been sufficient, in the past at least, to localize more or less successfully the lesions of tuberculosis. On the other hand, a deep, acutely-formed cavity with ragged sloughing walls of infiltrated tissues is not an evidence of immunity. On the contrary it is one of the proofs of a wholesale destruction of tissue and argues rather a total loss of resistance.

The large, firm-walled cavity is characterized by a deep-pitched and resonant percussion-note, by breath-sounds of a cavernous or amphoric quality as respects the fundamental tone and its overtones, but also with harsh and loud tones of higher pitch and bronchial quality. A prominent sign is râles, loud and consonating, of a variety of kinds and size, according to the viscidity and amount of the liquid contents of the cavity. Sometimes in advanced cases the mucus of the cavity is of a greenish yellow color, more or less bloodstained and of an extraordinary viscidity, so that it is expelled with difficulty. The viscidity which has been ascribed to the presence of nucleinic acid leads to a remarkable variety of râles; snoring, bubbling, creaking râles, the "sound of new leather," simulation of friction-rubs and the like, which disappear or are changed in quality after a coughing fit. Very characteristic of cavity is pectorilo-

quy, the voice appearing to proceed from the depths of the cavern and sometimes with an almost painful intensity. Whispered pectoriloquy, though less common, may also be heard. In a deep chronic cavity the only sign may be a deep and much prolonged expiration. Only deep-pitched sounds being conducted through air-bearing tissue, pectoriloquy and the bronchial tones of breathing are not to be heard. Exceptionally, however, the wall of the deep cavity is consolidated, in which case these cavity signs may be audible. Somewhat less rarely signs of cavity are

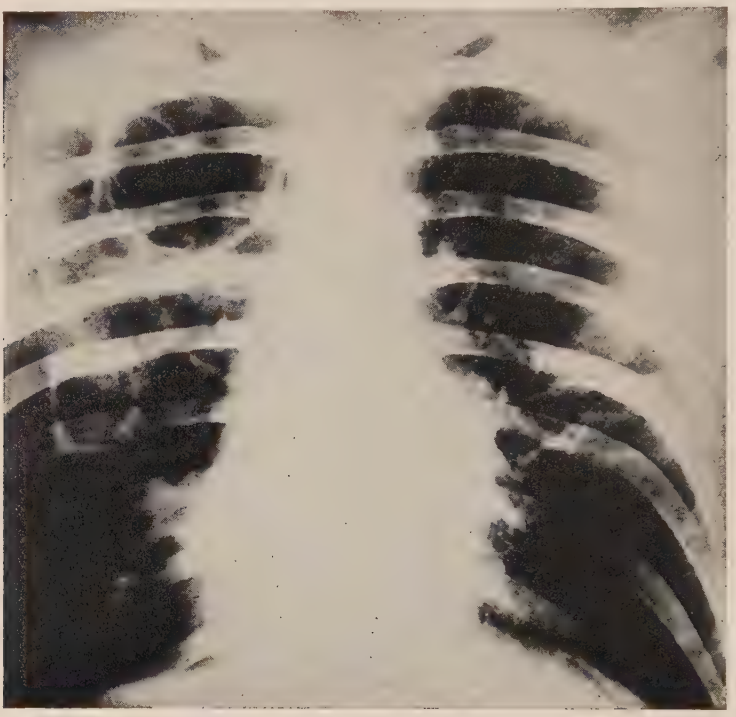


FIG. 35.—Large tuberculous cavity with thick wall in left lung, extending from first to fifth rib. Between the sixth and seventh ribs a second smaller but goodly sized cavity. The left lung has been partly collapsed by the introduction of air into the pleural sac (artificial pneumothorax). The heart is nearly tubular in shape (vertical dropped heart). Radiogram of chest seen from behind.

heard over a limited area of the chest wall. The question should then be asked: does the area of increased conduction correspond with the actual size of the cavity, or is conductivity especially good (as from localized caseation) over a portion only of the wall of a larger cavity? The latter is to be suspected if the cavity signs are typical, for such signs are only given by at least fairly good-sized cavities. The distinction is made of course by the pitch of the fundamental tone of the percussion-

note and of the breath-sounds; the lower the pitch, the larger the cavity. Observations of this kind, it may be pointed out, go to prove that the difference in physical signs, which seems so marked between the superficial and the deep cavity, depends in part at least upon the conductivity of the walls and not upon actual differences in the sounds produced within the cavity. This is true as respects pectoriloquy and bronchial breath-sounds, but the walls of the deep cavity can nevér have firmness and elasticity such as the ribs impart to the superficial cavity, so that the amphoric tones and the metallic phenomena are not to be expected in the former. Sometimes the walls of deep cavities are brought into apposition with one another after expiration and cough. Their separation in the following inspiration may then be accompanied by a whiff and by numerous sounds resembling moist râles. The method of expiration and cough should, however, be used very sparingly if at all in such advanced cases of pulmonary tuberculosis. A recent, or forming, cavity will be filled more or less with crumbling cheese and pus, so that the characteristic cavity sounds will be wanting or inconspicuous. The presence of such a cavity may be suspected, if it is superficial, from the presence of large consonating râles. A deep cavity in its formative stage will probably not be detected at all. Very small cavities can only be diagnosticated if superficial. The smallness of the volume of air forbids the formation of the amphoric tones and the resonant qualities of larger cavities will probably be absent. Usually what is diagnosticated in such a case is the presence of a lesion in which a cluster of consonating râles, unduly large for the location, create the suspicion of a small excavation. Cavities, large or small, if filled with fluid are equivalent to abscess and, except for dullness on percussion, give negative physical signs. Occasionally a closed cavity or abscess, filled with liquefied contents, is located so near the pleura that its weight drags upon that membrane and excites the characteristic, hopelessly unproductive "pleural reflex" cough which once heard will never be forgotten. A variety of the deep cavity is the serpiginous cavity, a narrow sinuous tube which extends sometimes deeply downward through the lung. If hemorrhage ensues in a cavity of this kind the blood can hardly be raised; the greater part of it infiltrates the surrounding lung. In hemorrhage into this form of cavity and indeed in all deep-seated pulmonary hemorrhages, the prognosis is exceedingly bad if the patient's face becomes cyanosed, though external hemorrhages have ceased or be slight.

If it should happen that all of the tubercles of a lesion were comprised in the caseated area of a tuberculous pneumonia, the evacuation of the cheesy mass would practically cure the lesion. It was thus that Robert

Koch first conceived of the cure of tuberculosis through tuberculin. But, unfortunately, as a rule, outlying tubercles persist which are not involved in the cavitation and, in any case, if the tuberculosis were the first tuberculous infection of the body that Koch assumed it to be, the tracheobronchial glands would also be contemporaneously involved. Cavitation is not, therefore, a curative process in the sense that it can by any possibility eliminate tuberculosis from the patient's body. It is, however, beneficial in that it effects the removal of enormous amounts of poisonous material.

FIG. 36.—Miliary tuberculosis. Illustrates the breaking down of caseations with the formation of cavities which have little, if any, fibrous encapsulation. Below, the lung is filled with miliary tubercles in the pneumonic stage. They project like knobs from the cut surface; hence the term "tubercle."

Miliary Tuberculosis.—The term "miliary" refers merely, of course, to the size of the tubercles. A better name would be disseminated hematogenous tuberculosis. The complete failure of immunity which so often immediately precedes and is the cause of death in tuberculosis, is the sig-

nal for the development everywhere of tubercle, often minute and only determinable by the microscope, sometimes of a more chronic type and appearing as macroscopic yellow tubercles. It is incorrect to speak of death as occurring from miliary tuberculosis in such a condition. The term should be reserved for cases in which there is a large or universal dissemination often from an old and apparently insignificant focus of minute tubercles, in the absence of large and active tuberculous foci in the lungs or elsewhere, the miliary tuberculosis thus constituting the principal manifestation of tuberculosis. Much study has been expended upon the causes of this condition, but the point upon which especial emphasis should be laid is that the initial condition which is the real cause of miliary tuberculosis is the complete failure of immunity which allows collections of tubercle bacilli to be set free and to establish everywhere new colonies. Miliary tuberculosis, as studied in the lungs, gives at the outset no signs and symptoms. On physical examination of the chest in a case in which the overwhelming of the vital forces is apparent day after day, no signs are obtained. At last distant and faint atypical râles are to be made out. By this time there is a characteristic restraint of the breathing. The lung expands slightly and ineffectually and on auscultation gives the impression of a body which moves slightly amid wet surroundings. If life is prolonged until softening of the tubercle has taken place the râles are more characteristic and nearer to the ear and the x-ray gives evidence of a multitude of minute shadows.

Hematogenous disseminations of tubercle may be sparse or dense. Their characteristic feature is their irregularity of dissemination, which is more apparent when the foci are distributed sparsely. Peribronchial tuberculosis is known at autopsy by the grouping of parenchymatous foci along the course of the bronchi and by "clover leaf" arrangement of tubercle on the cut surface which represent sections of bronchi and bronchioles filled with caseous material or with pus. When section at autopsy shows thickly-set tubercles, in great part of the same size and equally placed, which have invaded a whole lobe or the greater part of a lobe hitherto spared by the disease, the condition is not miliary tuberculosis, but the last stage of a bronchitis or bronchiolitis which is the termination of the more marked forms of peribronchial tuberculosis.

TUBERCULOSIS AND ARTHRITISM

Old age is characterized by an inability to assimilate food, there being an intolerance of many foods that were formerly well borne. This is

equivalent to saying that many poisons now accumulate within the body which formerly were eliminated or destroyed. Senility is not measured by years. It is the expression of a chronic intoxication. Intolerance of food may be inherited from ancestors who lived too well; it may be acquired prematurely by excesses in eating and drinking. A premature and partial senility of this kind is known as arthritism from the frequency with which the joints are involved. All human beings who live long enough become arthritic to some extent—progressively increasing accumulations of various poisons in time inevitably make themselves apparent. All old persons likewise become more or less tuberculous, or more accurately, the universal tuberculization manifests itself more distinctly in the old than in younger persons. Nevertheless arthritism prevails in the majority of cases over tuberculosis—the arthritic has a less severe tuberculosis than one who has no taint of arthritism. This is a fact that has never been adequately explained. There are, however, other instances of one disease protecting against another. The relation of arthritism to tuberculosis, relatively inconspicuous in youth, becomes increasingly important as the years advance. The peak of the mortality from tuberculosis per 1000 living is at about the 65th year; that is, of those who are alive in their 65th year a greater percentage will die of pulmonary tuberculosis than in any other year of life. After the age of 65 the ratio of deaths from pulmonary tuberculosis declines very rapidly. The high mortality at 65 is not, therefore, simply due to old age.

Old persons usually have more or less bronchitis; they have cough with a mucopurulent expectoration and the sputum very often contains tubercle bacilli. And, not very rarely, they have hemorrhages from the lungs. Such findings seem to make a clear enough diagnosis of pulmonary tuberculosis. It is customary to estimate their importance on the same basis as in the tuberculosis of the young, to take the tuberculosis seriously, to demand increased rest, life in the open air, abundant feeding and the like. No course could be more disastrous for the patient in the great majority of cases. Too often the treatment is more deadly than the disease, shortening a life that tuberculosis might have spared. It should always be remembered that in advancing years the arthritism remains almost always the predominant condition, so far as the complication with tuberculosis is concerned. Pulmonary tuberculosis, if any exists, is usually of the benign type—the patient is not much more than a bacillus-carrier. The bronchitis is not tuberculous by origin but metabolic, due to the elimination by the bronchial mucous membrane of poisons that should have been destroyed or otherwise eliminated. And the hemoptysis is probably not due to tuberculosis at all. Whatever the facts as to tuber-

culosis, the age of the patient leaves no doubt as to the existence of the arthritism; that is, of the accumulation of poisons which in one way or another will ultimately terminate life. That indication is, therefore, the first to be met; the patient probably does not need rest, but moderate exercise; not stuffing, but the lightest of foods and the best attainable elimination. And while there is no objection to fresh air in itself, the aged do not well support the vicissitudes of the out-door life and their bronchitis especially is made worse by draughts. A word as to the hemoptysis which is often the symptom that appears to clinch the diagnosis of tuberculosis. Arthritism is characterized, among many other signs, by a tendency to congestion, it may be of the roots of the teeth, or of the joints, or of the viscera. Such congestions (or temporary irritations of some kind) tend to occur in places of least resistance, as in tissues damaged by previous disease or injury. A congestion may lead to hemorrhage, especially if the blood-pressure is high. Such hemorrhages may take place from parts which are accessible to inspection and which are found by examination at the time or subsequently to have been to all intents and purposes normal. Reference is made here especially to epistaxis and to hemorrhage from the hard palate. From analogy we may conclude that it may be possible to have a hemorrhage in the arthritic which proceeds from practically normal bronchial mucous membrane as well as from one damaged by chronic inflammation, though not actively invaded by the tuberculous process. Hemoptysis of this kind is usually slight, but a larger hemorrhage, as an eliminative process, is not necessarily of gravity. As has been stated elsewhere, moist râles are obtainable for a week or more after the bleeding. In themselves they do not indicate anything more than the fact already known, that blood has been effused. If the hemorrhage was due to arthritism the bronchi will soon be found to be absolutely dry. Allowance must, however, of course be made for preëxisting bronchitis, bronchiectasis and the like. The location of the râles that follow hemorrhage is an important aid in discriminating tuberculous from arthritic hemorrhage. If the blood is raised easily and if the râles that follow its effusion are found in the upper part of the lung, it is safe to consider this region as the site of the hemorrhage. If it is also the site of an active tuberculous process the fact should be easily ascertainable by physical diagnosis. Hemorrhage raised with difficulty and presumably coming from the deep lung is, theoretically, more likely to come from an active tuberculous process; it is excessively rare except in the recognized consumptive. The cause of the hemorrhage can be determined with reasonable certainty by answering the question: do or do not the râles disappear within two weeks after the date of the

hemoptysis? The presence of tubercle bacilli in the sputum calls for increased care in the discrimination; it should not alter fundamentally the conception of the pathology of the case unless physical diagnosis shows by the usual signs that localized activity of a disease process is present somewhere in the lungs. That finding is of course to be duly respected. But the class which is now particularly considered, as the class most often misapprehended, is that in which the patients do not give evidence of an active localized tuberculous inflammation anywhere. Persons living in advanced age have survived their contemporaries; for one reason, because they have a high resistance to tuberculosis. One should not, therefore, be diverted from the treatment of the arthritism which is certainly present and which certainly, in the end, will be fatal, by hasty or routine assumptions of the predominance of indications for the treatment of tuberculosis when, in the absence of distinct evidences of localized tuberculous activity, this resistant class furnishes some of the classical signs of tuberculosis. A certain proportion of the aged, like those of lesser years, will die of a miliary tuberculosis which sometimes develops on the base of few and apparently obsolete tubercles. This fact should not lead to a reconsideration of the principles laid down above. For, as Fischer has shown, tuberculosis is often reactivated by a cachexia. It is then simply one of the indications of the complete breakdown of the patient's vitality before death. The fundamental indication here in the prophylaxis of tuberculosis is to treat or prevent the cachexia, in so far as this may be possible.

THE TUBERCULOSIS OF CHILDHOOD

It is necessary in taking up the diagnosis of the pulmonary tuberculosis of children to preface the study by as brief a consideration as possible of the pathology of tuberculosis of early periods of life.

The most simple and practical classification of pulmonary tuberculosis is based upon the result of the disease. The individual attacked by primary tuberculosis must literally conquer or die. If he conquers the result is a greater or lesser degree of immunization. If he dies the precise anatomical distribution of the generalized disease that carries him off is a matter of secondary importance.

The forms of the pulmonary tuberculosis of the child may be classified as to their outcome as follows:

Primary Tuberculosis of Very Young Children.—1. A small parenchymatous lesion and a small glandular lesion of one of the hilus or

tracheobronchial glands. Tuberculous lymphangitis of lymph-vessels which connect the two foci. Benign form.

2. A large parenchymatous focus with large caseous gland of the hilus. Miliary disseminations throughout the lung, generalized miliary tuberculosis; tuberculous meningitis. Fatal form.

Secondary Tuberculosis.[6] The Tuberculosis of the Earlier Stages of the Immunization of Older Children.—1. Tuberculosis lymphangitis proceeding from a gland or glands of the hilus perivascularly and peribronchially through the lower lobes, accompanied by a number of deeply situated discrete tubercles which especially involve lymphoid tissue, but as a rule without involvement of the superficial lung. Usually terminates in arrest. Lymph glands not much enlarged. Benign form.

2. Parenchymatous foci with little tendency to arrest or to localization. Disease often terminates in miliary tuberculosis and tuberculous meningitis. Groups of lymph glands may be moderately enlarged. Usually fatal form.

The arrested primary tuberculous process of early childhood has already been described in the first part of this chapter. On inspection of the foregoing table it will be seen that the most marked distinction between the primary and the secondary forms with bronchopneumonic foci is the great swelling of lymph glands in the one, its relative absence in the other. It will also be noted that the tuberculous process is benign so long as it remains confined to the lymphatic system, but that the primarily infected or but slightly immunized human organism cannot cope with extensive infections which have invaded the lung-parenchyma.

When the primary gland lesion is located in a tracheobronchial or hilus gland the lung bears the brunt of the attack of the disease. The region of the lung tributary to the primarily inflamed gland is first and most seriously involved. There may be a certain localization of the tuberculosis in the form of bronchopneumonic foci. In addition to such foci, the disease disseminates itself rapidly throughout the lung, other organs —especially the spleen and the liver—are contemporaneously involved and the disease runs a rapidly fatal course as a generalized tuberculosis. A general involvement of lymph glands is characteristeric of primary tuberculosis, but the enlargement is not usually great except in the case of the gland belonging to the region primarily infected.

Koch noted in his earliest experiments in inoculation of animals with tuberculosis that when a reinfection is made after an interval of several weeks or longer the glands of the part concerned in the inoculation do

[6] The term "secondary" is used here in contrast with "primary," but with no further implications.

not swell like the glands of the first inoculation or if they swell the swelling is temporary. Many later investigators have found that if the reinoculations are made too soon after the original infection this does not hold true—the glands do swell and the reinfecting dose is practically added, in its pathological effects, to the primary dose of tubercle bacilli. The glands, however, even in this case do not swell to the same extent as the primarily involved gland. These observations are most important for an understanding of the pathology and diagnosis of human tuberculosis.

Every human being, in our civilization at least, practically without exception must go through with a primary infection with tuberculosis. What the result of this infection will be depends upon various conditions; the size of the original infection, whether or not infections are frequently and too soon repeated and the health of the individual. If the original infection is small and if reinfections do not occur until some degree of immunity has been established, there is a primary "abortive" lesion which passes unnoticed and produces a degree of immunization such that, whatever the severity of later infections (exogenous or endogenous), the glands do not again swell to any material degree.

If the original infection is small enough to avoid a fatal primary tuberculosis but is followed by other reinfections at short intervals, the glands do not acquire so great an immunity. We have then the scrofulous type, of which chronically enlarged glands are a feature, but now the fulminant character of primary tuberculous adenitis is absent. The scrofulous type of childhood tuberculosis is capable of arrest. When the chief manifestations of scrofula are found in superficial glands the fact of their cure is easily ascertained and, it has been noted, leads to the establishment of a certain degree of immunization. The observed facts, so far as relates to cervical tuberculous adenitis are embodied in Marfan's law: "One almost never finds pulmonary tuberculosis, at least manifest and active pulmonary tuberculosis, in subjects who during infancy were attacked by scrofula (suppurative tuberculous adenitis of the neck) and who have completely recovered from it before the age of fifteen years, the cure having taken place before any other focus of tuberculosis has been appreciable."

Cases of pulmonary tuberculosis in which there are scars of the neck from a tuberculous adenitis are not very rare. Possibly the patients did not fulfil the conditions laid down by Marfan for the acquirement of immunity from subsequent attacks of pulmonary tuberculosis. However that may be, it is an important fact that even in the pulmonary tuberculosis of adults who have been scrofulous the glands of the thorax do not become materially enlarged during the progress of the lung disease.

The description which has been given of the course of a primary tuberculosis is applicable to *primary* tuberculosis of all ages. The disease is as rapid and malignant in the adult as in the child. This fact shows that the difference in type, which is usually so marked in adult pulmonary

FIG. 37.—Caseation of the bronchopulmonary lymph-nodes of the right lower lobe and of the lower and upper tracheobronchial lymph-nodes of the right lung. The corresponding lymph-nodes on the other side are free. Adhesive tuberculous pleuritis on the right side. Disseminated acute miliary tubercles in both lungs. The lungs of a nine months old child, viewed from the rear. (Ghon, "Der Primäre Lungenherd bei der Tuberkulose der Kinder," Urban and Schwarzenberg.)

tuberculosis as contrasted with the tuberculosis of infancy, is due, not to age, but to the ripening of a relative immunization against tuberculosis.

When tuberculosis has its most conspicuous lesions within the lungs it is customary to call the disease pulmonary tuberculosis. The pulmonary tuberculosis of the primary infection is not, however, comparable to the pulmonary tuberculosis of the adult, for it is not a localized tuberculosis.

In the pulmonary tuberculosis of older children the course of the disease, since it is less rapid than it is in infancy, may be said to manifest the influence of an incipient immunization. But the tuberculosis is still far from resembling the adult type and, while there are rare exceptions, it may be stated as the general rule that pulmonary tuberculosis of the adult type is not seen until adolescence is nearly past and that the manifest

tuberculosis of the younger child is generally a fatal malady. The type of the pulmonary tuberculosis depends, of course, upon the degree of immunization, not upon the age of the individual.

While the lesions of pulmonary tuberculosis of the adult almost always reach the surface of the lung, this is not so uniformly to be expected in the more acute bronchopneumonias of early tuberculosis. If the tuberculosis is deeply situated the diagnosis becomes correspondingly difficult. The larger lesions may be, and more frequently are, located in the lower lobes, the apices are not involved and the course of the disease in general differs greatly from the pulmonary tuberculosis of the adult. It is a mistake to expect to find in the child the characteristic signs of adult tuberculosis of the lungs.

In roentgenographs of ordinary cases of adult pulmonary tuberculosis and also of individuals who have never had manifest pulmonary tuberculosis, evidences of tuberculous infection of the lower lobes are found as straight streaks of opacity, representing no doubt bronchi and blood-vessels with thickened fibrous sheaths. These streaks, often densely aggregated, extend from the hilus region down into the lower lobes. With them are frequently associated dots and larger areas of dense opacity which can be no other than calcified glands and calcified parenchymatous tubercle, respectively. The streaks and the calcified dots are more numerous and more closely aggregated in, and, in fact, often seem confined to, the paravertebral portions of the lower lobe. Usually none of the lesions give evidence of having invaded the mobile base of the lung, their course and distribution illustrating the effect of the mobility of the part upon the development of tuberculous lesions which we have already considered. When discovered in the adult these lower lobe lesions are completely arrested. The region in which they are seen is not, as a rule, invaded by the processes of manifest pulmonary tuberculosis until comparatively late in the course of the disease. The lesions therefore contribute nothing to the early extensions of tuberculosis of the ordinary upper lobe type.

Analyzing the X-ray picture, we see that, as in primary lymphangitic tuberculosis, there is an especially marked fibrous thickening of the tissues about the large vessels and air-tubes at the root of the lung, to which—as the process extends out into the parenchyma of the lung—there succeed fibrous thickenings, no longer fused into a single opacity but plainly connected with the outer coats of vessels and tubes. The pathological picture agrees, therefore, in the two cases both as respects the relatively more marked inflammation of the hilus region and as to the tuberculous lymphangitis of the deep-lying bronchi, but the later process

is enormously more extensive than the early process. The most striking difference between the two conditions is in the mode of termination of the deep extensions of the lymphangitis. In the primary type the disease process regularly extends to the superficial lung to terminate in a parenchymatous tubercle. In the secondary type, on the other hand, the lymphangitis regularly aborts in the deep lung or terminates in a deep tubercle. One can hardly conceive that the extensive peribronchial inflammation of the secondary process could have taken its inception in aërogenus infections of the deep lung, such as have been assumed in order to account for the simpler primary process; the similarity of the two types in other respects justifies the belief that the primary process also begins at the hilus and works outward.

It is customary to ascribe bronchovesicular breathing and a percussion-note higher in pitch and shorter than the normal in the interscapular region to the influence upon the physical signs of relatively superficial bronchi and to consider these signs as normal for the location. By some writers the breathing of the interscapular region is said to be mixed breathing. But the simultaneous presence, under the bell of the stethoscope, of normal and bronchovesicular breathing shows abrupt changes in the conductivity of pulmonary tissues, which are more likely to be due to pathological causes than to normal anatomical relations. And, while the signs mentioned are very frequently found, they are sometimes absent in chests which do not differ in configuration from others that show them well. The writer is, therefore, of the opinion that they are usually to be brought into relation with a healed hilus tuberculosis.

As we have seen, primary tuberculous processes are sharply distinguished from secondary processes by the behavior of the glands. Acutely swollen glands are characteristic of primary tuberculosis and, therefore, in view of the early date at which tuberculous infection usually takes place they are only to be expected in very young children. Hamburger states that it is rare that large swellings of lymph glands are found later than the second year. These distinctions as to age and the influence of immunization upon adenitis have not generally been taken into account, and glandular swellings have been believed to dominate the pathology and the physical signs of the hilus at ages in which such swellings are, to say the least, rare.

Enlarged tracheobronchial and hilus glands may cause death by rupture into veins and bronchi without symptoms that indicate their existence. In some cases, however, they produce pressure upon neighboring organs, the symptoms naturally varying according to the location of the gland and its size. These symptoms are: dyspnea which may be either ex-

piratory or inspiratory, cyanosis from pressure on large venous trunks and cough of varying type, being sometimes spasmodic, sometimes continuing for weeks without cessation.

Eustace Smith's Sign of Bronchial Adenopathy.—If the head of the child is bent backwards so that the face becomes horizontal and the eyes look toward the ceiling, a venous hum is sometimes heard over the manubrium, which becomes less loud when the chin is depressed and ceases shortly after the head resumes its normal position. The sound is explained as due to compression of the left innominate vein, as it passes behind the sternum, by the glands at the bifurcation of the trachea. This sign is not regarded as very reliable. It is said to be sometimes found in short-necked children who have no enlarged glands.

D'Espine's Sign.—This sign has been considered to be of considerable value in the diagnosis of enlarged tracheal glands in the adult. It consists in the auscultation of the voice, or preferably the whisper, over the spines of the vertebrae. In the adult the transmission of the voice is good, normally, until the fourth dorsal spine is reached, as one proceeds downward listening over the spinous processes in succession. Exaggerated transmission of voice or whisper over the fourth dorsal spine is considered to be pathological and the same over the fifth and sixth dorsal spines to be frankly positive for adenopathy. In the infant, on account of the higher level of the bifurcation of the trachea in relation to the spinal column in the earliest years of life, bronchophony over even the seventh cervical and first dorsal spines is thought to have a pathological significance. Since the patient does not render assistance to the examiner the auscultation for d'Espine's sign is more difficult in the infant than in the adult. It is, however, a sign of much greater value in the infant for it may in truth reveal the existence of a much enlarged gland or group of glands. While d'Espine's sign is of "pathological significance" in the adult, it will at once occur to the reader that the significance in the vast majority of cases can hardly be the presence of an adenopathy at an age when great enlargement of intrathoracic glands is a phenomenon of the greatest rarity in tuberculosis. The indubitable presence of much enlarged glands of the thorax in the adult case of pulmonary tuberculosis should lead one to think of Hodgkin's disease or other conditions, distinct from tuberculosis, in which enlarged glands are a feature.

The interposition of cartilaginous disks, as heterogeneous sound media, between the bodies of the individual vertebrae, as has already been pointed out, isolates each bone to a considerable extent, so that there is little transmission of sound from one vertebra to the one immediately succeeding it. Hence abnormal transmission of sound to the vertebra

examined means abnormal conductivity of sound to that vertebra from the region that lies directly before it. Such improvement of conductivity is generally ascribed to conduction of sound from the trachea by an enlarged gland, at whatever age it may be detected. There is no reason, however, why condensation of tissue from fibrosis or inflammatory infiltration should not also produce such a result. But, according to Barlow and Thompson, d'Espine's sign in the adult is always due to the presence of a partial pneumothorax. It would be expected that if the tracheal sounds are transmitted through a volume of air as through a resonator they would be more distinct and have a more clearly defined pitch (fundamental note of the cavity) than when the transmission is through solids. In the present state of our knowledge all that can be claimed for d'Espine's sign is that increased conduction of voice-sounds over the vertebrae at the locations indicated is proof of an abnormality of the tissues before the spine which, in the great majority of cases, is due to tuberculosis. But even then the sign is to be received with caution as evidence of an active process, for the more mature the fibrosis the better it conducts sound. In general, after the first years of life it is a mistake, in the absence of very clear indications, to ascribe dullness on percussion and changes in breath-sounds and in the transmission of the voice in the interscapular region to enlarged glands rather than to extraglandular inflammation, without of course necessarily excluding the presence of moderately enlarged glands in the inflamed tissues.

The physical signs of the arrested tuberculosis of the hilus region proceed from the fibrous thickenings which the x-ray so clearly shows. We are really investigating the central end of the lymphangitic process which, commencing with the gland, stretches out toward the periphery in a manner analogous to the primary lymphangitis, as well as to the abortive tuberculosis of the upper lobes.

The diagnosis of the deep-seated extensions into the parenchyma from the hilus has hitherto practically only been made by the x-ray after the arrest of the process. The depth at which the lesions are situated will always make the physical diagnosis difficult but it is probable that, if the attention of the diagnostician were concentrated upon the paravertebral portion of the lower lobes and distant and ill-defined râles were sought for, as in the diagnosis of other peribronchial tuberculosis, the process might be detected while it is still active.

One of the chiefest difficulties in the diagnosis of the obscurer manifestations of the tuberculosis of childhood is that of obtaining the opportunity to examine the child when the disease is active. It is not uncommon for previously healthy children to have periods of unexplained ill

health. They lose their appetite, become listless and fretful, have a slight fever, etc. There are, of course, many other possibilities of infection under such conditions which should be considered, but an examination of the interscapular region for evidences of tuberculosis should not be neglected. Because of the resonance of the child's chest, percussion should be light. The second and third intercostal spaces should be examined with especial care. Symmetrical points on the two sides should be compared with one another and possible differences in the note at different levels should be determined. Distinctly marked and localized differences in the percussion-note, however slight, are more important in the diagnosis than the supposed determination that the whole region is, or is not, duller than it should be. Auscultation of the breath-sounds should be carefully made with the view to determine shades of difference in the breathing over different points. Here again, local differences, even the slightest, especially if abrupt, are of more significance than the determination of a normal or abnormal intensity of breath-sounds over the whole region examined. The front of the chest should also be examined by percussion with especial view to the determination of dullness from enlargement of anterior mediastinal glands along the right edge of the sternum. Results from auscultation of this region are more dubious on account of the normally bronchovesicular character of respiration in this place.

In the study of these cases the x-ray is of great value. An enlarged gland, or group of glands, if not too recent, may be shown by the roentgenograph. Calcifications are, of course, not significant of present trouble. Their presence speaks, rather, against the diagnosis of active inflammation, for a healed tuberculosis does not readily recrudesce in its original site. In well-marked cases of tuberculous activity a triangular shadow with base toward the mediastinum may be seen to extend into the lungs from the hilus. According to Sluka, this shadow is never found in the two first years of life, which fact shows the phenomenon to be associated with the inflammatory reaction of secondary tuberculosis. The x-ray negative may also show cloudy masses with irregular outlines, due to the involvement of the parenchyma in more chronic bronchopneumonic processes. Both lungs are usually involved in this type of tuberculosis, one side often to a much greater extent than the other. The x-ray is of the less assistance the more recent the process. A negative finding does not, therefore, exclude hilus tuberculosis; the x-ray examination in that case should be repeated after an interval. And it should also be repeated notwithstanding an apparent complete recovery of the patient. For it should be remembered that the group of symptoms

which has called attention to the case, if they were of tuberculous origin, probably developed at the time of an exacerbation of the same character as that of the more superficial manifestations of tuberculosis. The child felt badly because he was having a reaction to tuberculous poison. After the reaction has passed he should be reëxamined for a further determination of the case. A roentgenograph may now show sharper lines and diminution or absence of diffuse shadows. This is encouraging but it does not, of course, prove that the original diagnosis was wrong nor that hygienic care is not necessary. A serious and not uncommon mistake is to regard evidences of a past and obsolete tuberculosis as proof of active inflammation. The distinction is chiefly made by the sharpness of outlines. There is usually uniform shadow in the immediate vicinity of the hilus, even in healed cases, but in such cases shadows should not melt imperceptibly into the clear lung-field and there should be no fuzziness of bronchial trunks. Sharply marked bronchial outlines undoubtedly mean a mature fibrosis and an old lesion. A healed parenchymatous lesion of fair size well very likely persist in the x-ray picture as a more or less diffuse opacity. The presence of calcified lymphoid tissue at the bifurcations of bronchi afferent to the affected region and, still more, calcifications within the opaque region itself discloses the age of the process.

The von Pirquet cutaneous tuberculin test is of considerable value in the diagnosis of the tuberculosis of infants at the age when the occurrence of primary infection is still doubtful, but is of little value in older children in whom a positive reaction is not necessarily connected with the condition supposed to indicate tuberculosis. A negative reaction in children, however, usually, but not necessarily, indicates that infection with tuberculosis has not yet occurred. In such a case the child should be watched with especial care in order to ensure that everything is done to give a favorable course to the primary tuberculosis which may declare itself at any time. The subcutaneous use of tuberculin in these cases should not be considered for one moment.

PHYSICAL DIAGNOSIS OF DISEASES OF THE CHEST

THE HEART

CHAPTER I

NORMAL AND PATHOLOGICAL PHYSIOLOGY IN THEIR RELATION TO THE PHYSICAL DIAGNOSIS OF DISEASES OF THE HEART

Introduction

The heart is a part of the whole circulatory system and it should be considered as such, rather than as an independent organ. Its close relation to the arteries is shown by its development in the embryo from a twisted arterial tube. The respiratory system must also be investigated in studying the heart, as usually the first indication of weakness of the heart is difficulty in breathing on exertion. The functions of the kidneys are closely related to the heart and a diminished secretion of urine is a frequent result of impaired circulation of the blood due to cardiac weakness. Albuminuria and the presence of casts in the urine may be due to this cause. Digestive and nervous disturbances may be caused by failure of the weakened heart to send a proper supply of blood to the stomach, intestines, or brain.

Impairment of the power of the heart to do its work may manifest itself, at first, in subjective symptoms, such as a sense of suffocation or of forcible heart action, when the physical examination of the heart and other organs reveals nothing amiss. Hence, it is evident that, to determine the condition of the heart, the examination must embrace the subjective symptoms of the patient as well as the physical state of his organs.

The methods of physical diagnosis are those already described in dealing with disease of the lungs,—inspection, palpation, percussion and auscultation. These, together with a carefully taken history of the subjective symptoms, are the physician's chief reliance in obtaining trustworthy knowledge of the functional condition of the heart and circulation and of the pathological anatomy it presents. Other methods of investigation, such as the use of the sphygmomanometer, the orthodiagraph, electrocardiograph, the polygraph, and the spirometer, sometimes yield information of value not otherwise obtained, but they are accessory and supplementary to the ordinary clinical methods of physical examination and should be regarded as such. They should never be substituted

for the older and more valuable procedures which are available to every physician.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

Position of the Heart.—Three-fifths of the heart are to the left of the median line, and two-fifths to the right of the median line. The heart lies in the anterior mediastinum, but only a small portion is in contact

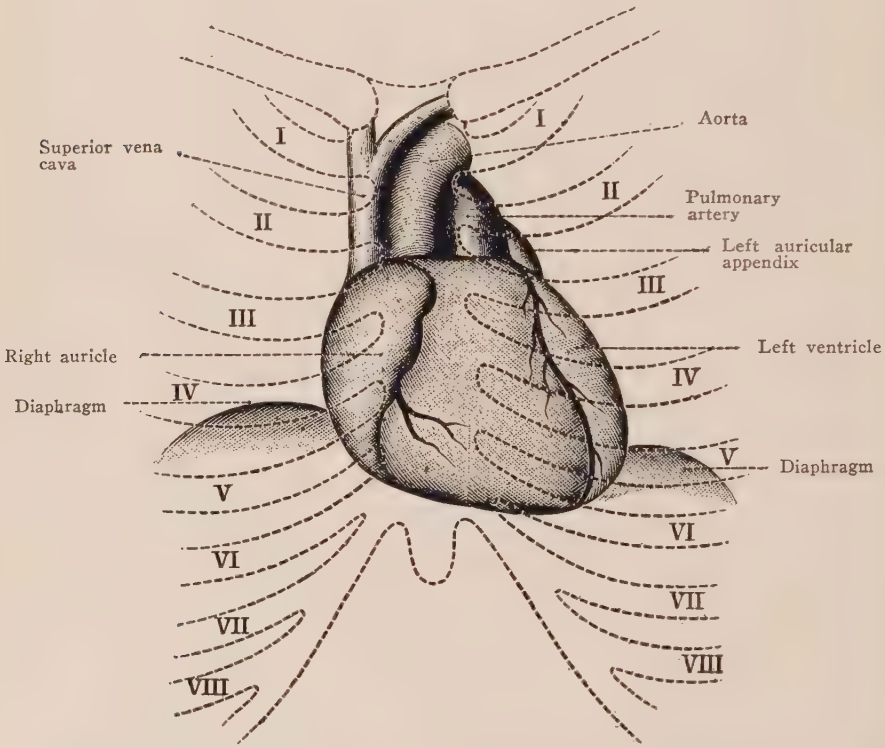


FIG. 1.—The position of the heart viewed from the front. Note that the right border of the heart is formed by the right auricle and the right border of the vascular attachment above by the superior vena cava. The left border of the heart and great vessels is formed by the left ventricle below; above this are the left auricular appendix, the pulmonary artery and the arch of the aorta. (Moritz, Deutsche Klinik, Berl. & Wien, IV.)

with the anterior chest wall. This area is behind the lower part of the sternum to the left of the median line and the fourth and fifth left interspaces near the sternum. The greater part of the anterior surface of the heart is covered by pleura and lungs. The portion of the heart in direct contact with the chest is decreased during inspiration by the movement

of the lung margins into the pleural sinuses, in front of the heart. The heart rests in a depression in the central part of the diaphragm and is supported above by the great vessels (vena cava superior, aorta, arteria pulmonalis). The lower surface is formed largely by the right ventricle. (Fig. 1.)

Knowledge of the topography of the heart is important and by means of the orthodiagram, it can be studied in the living. The right border of the heart is formed by the right auricle and not the right ventricle, as many students think. This right border in health extends upward in nearly a straight line to join the outer border of the superior vena cava, and it can well be seen in a radiogram of the heart or on the fluoroscopic screen. When the right auricle is dilated, the right border of the heart forms a convex arc which bulges some distance to the right of the line

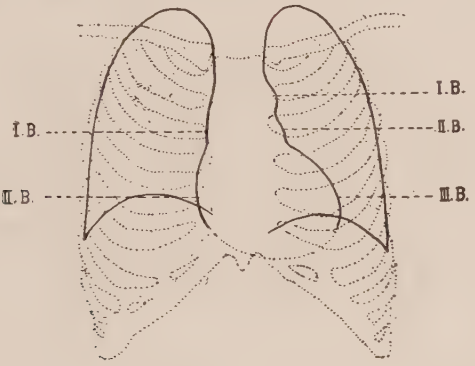


Fig. 2.—The arcs of the middle shadow of the thorax, seen in dorsoventral illumination by the Roentgen rays. Normal picture. On the right side of the heart the upper flattened arc is the superior vena cava (I.B). This joins the lower arc (II.B), formed by the right auricle, at the level of the third rib. On the left border is seen the projection formed by the aorta above (I.B.); below is the pulmonary artery (II.B.), and the prominent bulging of the left ventricle which extends from the third to the sixth rib. (Morawitz, "Klinische Diagnostik innerer Krankheiten," F. C. W. Vogel.)

above formed by the superior vena cava. The left border of the heart makes a gradual curve outward as it descends to join the lower border and form the apex. The apex is seen through the fluoroscope as a rounded outline. The left border of the heart, together with that of the great vessels, normally produces a contour, on the fluoroscopic screen, of three convex projections; the lowest and largest of these bulgings is caused by the left ventricle; the middle is formed by the pulmonary artery and the left auricular appendix; and the highest of the three arcs is produced by the arch of the aorta (see Fig. 2).

The heart extends from behind downward and forward and to the left.

The axis of the long diameter of the heart forms an angle of about 60 degrees with the long axis of the body, but this varies with the shape of the chest and during respiration. As the diaphragm rises the position of the heart becomes more horizontal.

Architecture of the Heart Muscle.—The heart muscle in the embryo is made up of a single muscle band, one end of which is attached to the

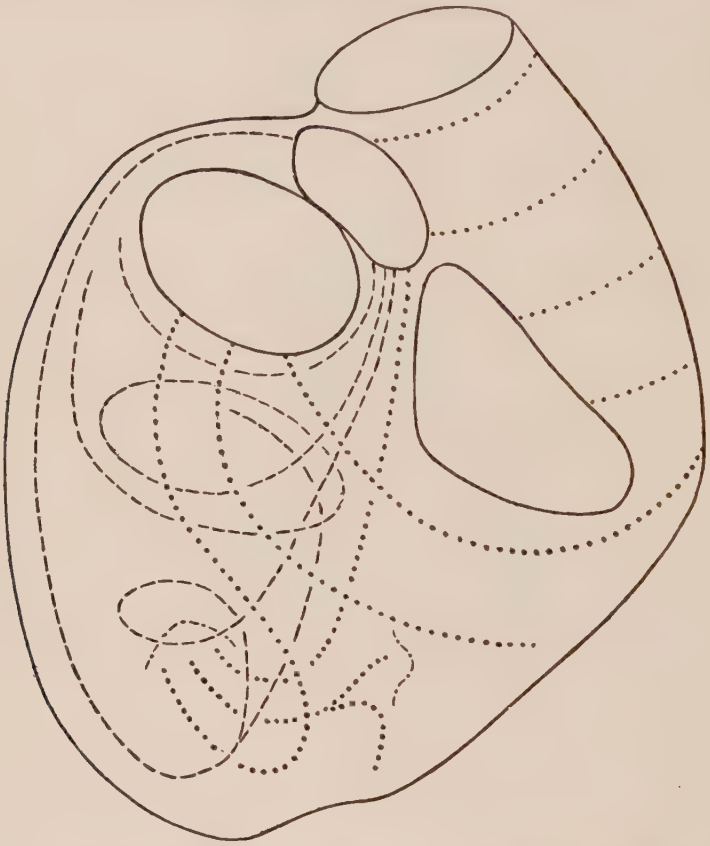


FIG. 3.—The chief muscle bundles are shown here. The bulbospiral group of fibres is represented by dashes, and the sinospiral by dots. The bundles immediately around the left ostium and the conus form single loops which attach themselves to the aortic septum. All other bundles may be considered modifications of these two simple loops. (Mall, Am. J. Anat., Balt.)

fibrotendinous ring that separates the auricle from the ventricle, the other end to the tendons of the papillary muscles. In the adult heart the arrangement of the muscle fibres is complicated, but they can be divided into four groups: (1) The superficial bulbospiral fibres; (2) the superficial sinospiral fibres; (3) The deep bulbospiral fibres; (4) The deep sinospiral fibres. (Mall.) Like other muscles they have tendinous at-

tachments. These tendinous attachments arise from the fibrous structures that surround the auriculoventricular orifices, the conus arteriosus and the aorta, and they end in the papillary muscles and the septum of the ventricles. The general direction of the superficial fibres is from above downward and to the left, running in a spiral curve, and when the apex is reached they form a vortex and pass upward into the interior of the heart. One set of deep fibres encircles the left ventricle, and another set surrounds the right ventricle. In the upper part of the ventricles the muscle is arranged in circular bands, especially well marked around the auriculoventricular and arterial orifices. Contraction of these ring muscles aids the valves in closing the openings they are made to guard. (Fig.

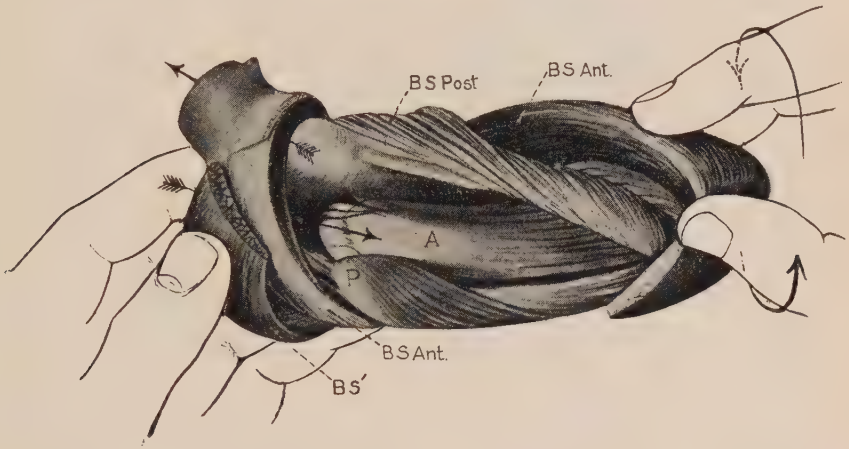


Fig. 4.—Pig's heart with the outer fibres removed to show the bundles within the heart. A and P, anterior and posterior papillary muscles, BS', portion of the bulbospiral band which encircles the left ostium; BS ant., portion of the superficial bulbospiral band which is attached directly to the root of the aorta; BS post., portion of the superficial bulbospiral band which encircles the heart in front and is finally inserted in the posterior ligament of the aorta. The heart is in diastole as the position of the left hand holding the apex indicates. (Mall, Am. J. Anat., Balt.)

3.) The arrangement of the musculature of the ventricles is such that when the heart contracts in systole it does so with a twisting motion similar to that given to a wet cloth when water is wrung from it (Mall). (Figs. 4 and 5.) The left ventricle empties itself by diminishing its transverse diameter without shortening the long diameter. The apex is drawn forward and to the right due to the contraction of the spirally arranged muscles. The right ventricle expels its contents by drawing in its external wall toward the septum and by shortening the length of the ventricle. This shortening movement is the cause of the descent of the

conus arteriosus during systole, so evident on inspection of the exposed heart.

The Valves and their Closure.—The auriculoventricular valves are delicate but firm structures with thin edges. They are attached at their base to a strong ring of muscle and connective tissue. Attached to their free margin on their ventricular surface are delicate tendinous cords which run to the tips of the papillary muscles. When the valves close their edges overlap. The orifice, which the valves must cover, is diminished in size during systole of the ventricle by contraction of the surrounding ring muscle. The papillary muscles, contracting synchronously

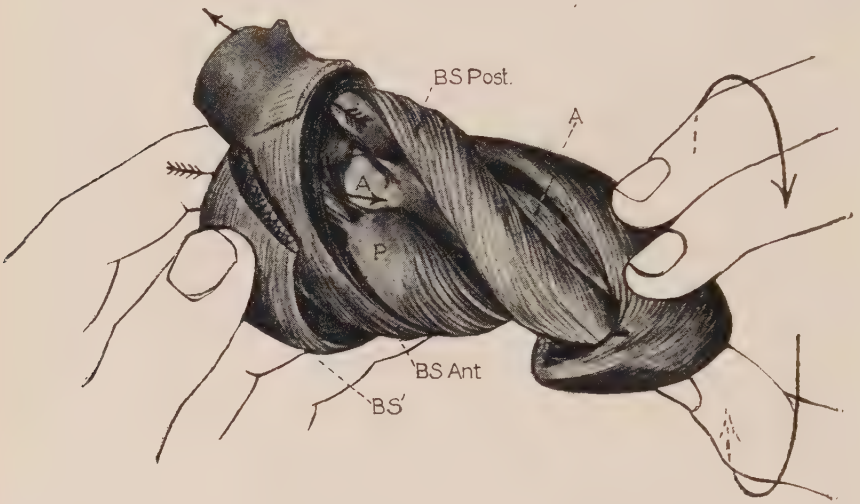


FIG. 5.—Same as Fig. 4, except the hand at the apex is twisting the heart to imitate the rotation which takes place when the ventricles contract. This action twists the inner muscle bundles and thus "wrings" the blood out of the heart. (Mall, Am. J. Anat., Balt.)

with the muscular wall of the ventricles, pull on the chordae tendinnae and prevent the rising pressure in the ventricle from swinging the surface of the leaflets backward into the auricle. This mechanism is a safety device against regurgitation of blood from ventricle into the auricle. The semilunar valves are denser and stronger than the atrioventricular segments. They are attached to a strong ring of muscle and fibrous tissue. When open, the atrioventricular valves are not in contact with the side of the ventricle but float in the blood stream. It has been generally held that they are pushed into the position of closure at the end of the auricular systole by the formation of eddies of blood between them and the ventricular wall. A more recent view is that they are closed by the movement

of blood from the sides of the ventricular cavity toward the center when the flow of blood from auricle to ventricle suddenly ceases at the end of auricular systole. The breaking of the jet leaves an area of negative pressure much as in the wake of a ship. From each side blood is drawn in and the valves are unrolled and approximated. (Henderson and Johnson.) A moment later they are closed effectively by back pressure in early ventricular systole. (Dean, Jr.) Similar explanations are given for the shutting of the aortic and pulmonary valves. The closure of the semilunar valves occurs, without jar or noise, at the moment the outflow

FIG. 6.—Diagram of a generalized type of vertebrate heart—combining features found in the eel, dog-fish and frog—showing the primary cardiac chambers and their lines of union. a, sinus venosus and veins; b, auricular canal; c, auricle, d, ventricle; e, bulbus cordis; f, aorta; 1–1, sinocanalar junction and venous valves; 2–2, canalo-auricular junction; 3–3, annular part of auricle, containing special muscle fibres; 4–4, invaginated part of auricle; 5, bulboventricular junction. By the longtitudinal fibres lining the ventricle there is a connection between the annular fibres of the auricle and the bulbus musculature. (Keith and Flack, J. Anat. & Physiol.)

ceases and the ventricles begin to expand. "The heart, as a good pump should, works with the least possible jar." (Flack and Hill.)

The Sinus Node.—The impulse which normally causes the heart to beat has its origin in a small mass of specialized muscle tissue called the sino-auricular node, or the Keith-Flack node. The tissue of which it is composed is the remains of the sinus venosus of the embryonic heart. (Fig. 6.) The sinus venosus persists unchanged in the adult heart of amphibia. This node is the pacemaker of the heart, and is situated in

the wall of the right auricle at its junction with the superior vena cava. In the human heart it is about the size of a grain of wheat. (Fig. 7.) Microscopic examination shows it to consist of pale cardiac muscle fibres with which nerve fibres appear to be in actual continuity. The peculiar muscle cells of which it is composed are similar to those long known to exist in the ventricles of the heart and called after their discoverer Purkinje cells.

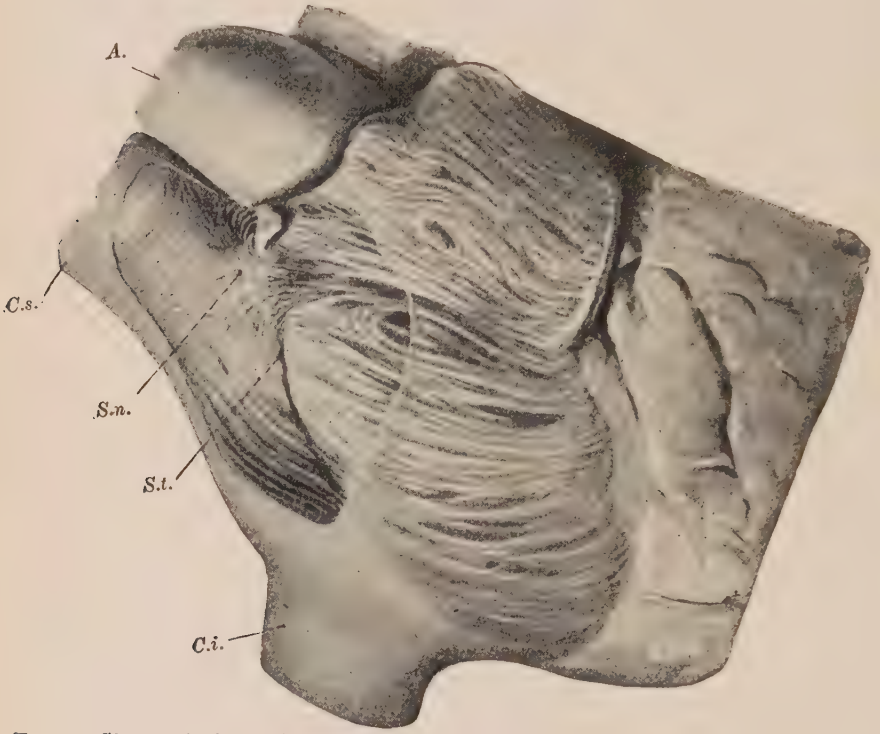


FIG. 7.—Sino-auricular node, represented macroscopically. The epicardium has been removed. Natural size. *A*, aorta. *C.i.*, inferior vena cava; *C.s.* superior vena cava; *S.n.* sino-auricular node; *S.t.* sulcus terminalis with the blood-vessel that lies in it. (Tandler, "Anatomie des Herzens," G. Fischer.)

The Conducting System.—The impulse to contraction originates in the sinus node, usually in its wider part, and spreads over the auricular muscle in a widening circle. The rate of speed is approximately 1000 mm. per second. This wave of excitation reaches a second node similar to the sinus node situated at the base of the interauricular septum on the right side, and below and to the right of the cavernous sinus. This is the auriculoventricular or the Aschoff-Tawara node. (Fig. 8.) It is in close muscular connection with the interauricular septum and thus indirectly with the sino-auricular node. From the lower part of the Aschoff-

Tawara node extends a bundle of conducting fibres which lie on the top of the interventricular septum below the pars membranacea septi. This is His's bundle and *it forms the sole muscular connection between the auricles and the ventricles.* The main bundle is about 1 cm. long, and divides below into a right and a left branch which pass to the right and left ventricles. The right branch is cord-like and is imbedded in the septum

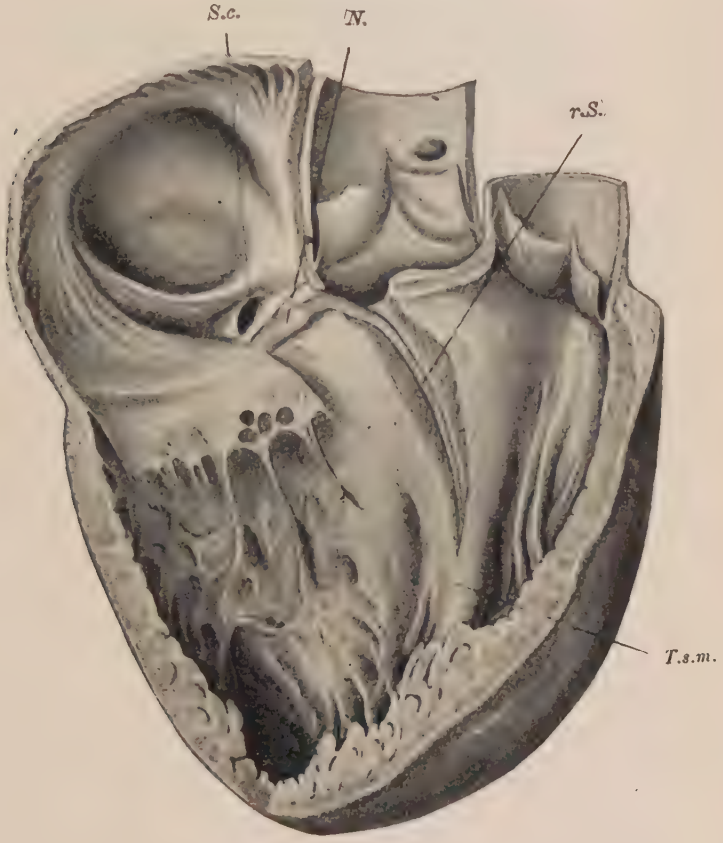


FIG. 8.—Auriculoventricular bundle and its right branch. Five-sixths the natural size. *N*, the auriculoventricular node of Tawara; *r.S.* the right branch; *S.c.*, sinus coronarius; *T.s.m.*, trabecula septomarginalis. (Tandler, "Anatomie des Herzens," G. Fischer.)

during the first part of its course. The left branch runs in the subendocardial tissue. (Fig. 9.) These main branches divide into many subdivisions which finally end in arborisations between the ordinary contractile muscle fibres. (Fig. 10.) Ramifications of the conducting system may pass across the cavity of the ventricle, from one wall of the ventricle to the wall on the opposite side. This regularly occurs in some of the

lower animals. In the sheep is normally found such a free bundle of conducting fibres. It is called the moderator band.

The muscle cells of the conducting system are smaller than the regular cardiac muscle fibres, strain less intensely, and resemble embryonic muscle cells. They are poorer in fibrils but richer in protoplasm and

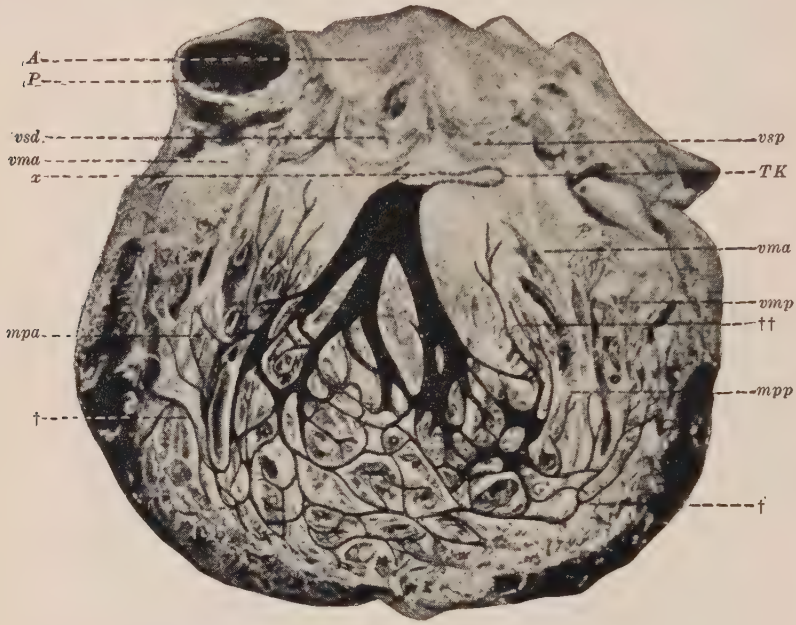


FIG. 9.—Human heart, left branch of the conducting bundle (after Tawara). *A*, aorta; *P*, pulmonary artery; *vsd*, right cusp of aortic valve; *vsp*, posterior cusp of aortic valve; *mpa*, anterior papillary muscle; *mpp*, posterior papillary muscle; *vma;* anterior segment of the mitral valve; *vmp;* posterior segment of the mitral valve; *Tk*, Tawara's node; *x*, point of division of the auriculoventricular bundle (His's bundle) into right and left branches; *t*, terminal ramifications of the left branch; *tt*, string-like strand crossing the cavity of the left ventricle and containing a subdivision of the left branch. (Morawitz, "Klinische Diagnostik innerer Krankheiten," F. C. W. Vogel.)

glycogen than the ordinary oval, faintly straining nulei, and can always be recognized by the abundance of their sarcoplasm which separates its myofibrils or fibrillar columns. (Van der Stricht and Todd.)

If the sinus node is destroyed or if its function is abolished by cooling, then the auriculoventricular node initiates the impulse that leads to contraction. Its automatic power can become active only after the sinus node ceases to act. If the auriculoventricular bundle is cut or destroyed then the ventricles beat very slowly. The impulse that gives rise to their contraction originates in the ventricular portion of the conducting sys-

tem. The automatic property is less in the secondary center, the auriculoventricular node, than in the primary center, the sinus node, and is still less in the tertiary centers situated in that part of the conducting system lying in the ventricular walls.

Properties of the Heart Muscle.—If the heart muscle responds at all to a stimulus it responds with a maximum contraction. This does not

Fig. 10.—Photograph of model of sinoventricular system in calf's heart. (De Witt, Anat. Record, III.)

mean that a weak, poorly nourished muscle yields as forcible a contraction as a well nourished, strong, muscle. It gives, however, the maximum contraction of which it is capable at that particular moment. Thus it differs from a skeletal muscle which gives a weak contraction when weakly stimulated and a strong contraction when strongly stimulated. Recent work has shown that the individual fibres of skeletal muscle obey the "all or none law" and that gradations in the extent of contraction of these muscles are due to the number of fibres that are excited. (F. H. Pratt.)

A property of the heart muscle is its transitory loss of excitability and for understanding certain disturbances of the heart rhythm, it is important to remember it. This period of inexcitability is known as the refractory period of the heartbeat. It begins when the excitation wave enters the muscle and lasts during the entire systole. After that the excitability rapidly returns. Conduction is also depressed after an impulse has passed through the conducting tissues. During this period the muscle will not respond either to the natural automatically generated heart stimulus or

to artificial stimuli. During diastole there is a gradual return of irritability.

The rate of conduction is rapid through the fibres of the conducting system, but the passage of the impulse is slow through the ordinary contracting fibres. There is an interval from one-fifth to one-tenth of a second between the beginning of the auricular systole and the onset of the ventricular systole. Apparently the impulse is held up for a short interval in the auriculoventricular node. Injury to His's bundle will cause a marked delay in the onset of ventricular systole.

Heart Dynamics.—The dynamics of the heart are similar to those of the skeletal muscles. If a strip of muscle is suspended from one end and to the other end is attached a weight the relaxed muscle will be extended by the pull of the weight to a certain length. Stretching the muscle with the weight temporarily changes the physical properties of the muscle so that its contractile power is increased. The extension of the muscle will cease when the tension developed equals the weight of the attached load. With reference to the later contraction of the muscle the tension that has been acquired by the muscle is called the *initial tension.* When the relaxed heart muscle is distended by blood during diastole the same change in the contractile power of the muscle occurs. The blood filling the ventricle plays the same part as the weight attached to the strip of muscle in the experiment described above. The heart muscle when stretched develops an initial tension. The greater the filling of blood within physiological limits the greater the initial tension. If a skeletal muscle extended by a weight to a certain length is prevented from shortening when stimulated, then the stimulus acts not by shortening the muscle, which it cannot do as the ends are fixed, but by increasing the tension above the initial tension. The muscle makes a so-called isometric contraction. Its tension is increased but its length remains unchanged. In the case of the left ventricle distended with blood at the end of diastole the stimulus to contraction cannot shorten the muscle because both the aortic and mitral valves are closed, but it does increase the tension. The stimulus gives rise to an isometric contraction. When the increase of pressure in the closed ventricle produced by the isometric contraction of the ventricular muscle exceeds that in the aorta, the aortic valves fly open and the muscle begins to shorten, expelling the blood from the ventricle. This is practically an isotonic contraction in which there is a shortening of the muscle accompanied by little change of tension.

The mode of contraction of the heart muscle can be imitated by a voluntary muscle when it raises a weight that is supported by a rachet during relaxation. (See Fig. 11.) During relaxation the tension is

determined by the length of the muscle. When stimulated the tension increases until it is able to raise the weight. This is an isometric contraction. The tension, when it exceeds the downward pull of the load on the rachet, causes the muscle to shorten. This is an isotonic contraction.

The more a skeletal muscle is lengthened by stretching within physiological limits the greater the tension that develops and the greater the force of the contraction on stimulation. This is true also of the heart

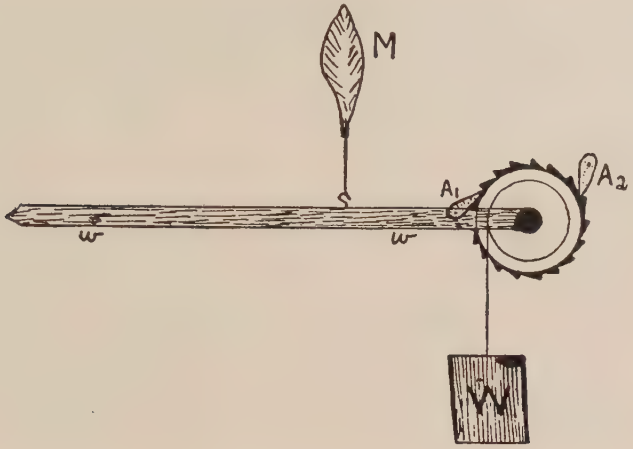


FIG. 11.—The muscle M after contraction is extended simply by the weight of the lever w. When it begins to contract, the arm on the lever catches in the ratchet wheel so that the weight W is drawn up, W being supported by the arm A2, so that the muscle after excitation cannot raise the weight W until its tension is equal to that of the weight. As soon as it has completed its contraction and begins to relax, the weight W is taken off it (cf. closure of aortic valves) and it is then extended by the small weight of the lever (pressure of inflowing blood). It is evident that the length of the muscle before it begins to contract depends on the weight of the lever, i. e. on the diastolic load. The extent to which it will contract depends on the tension aroused in it when it contracts and the amount of the weight W which it has to overcome. (Patterson, Piper and Starling, J. Physiol., XLVIII.)

muscle. The greater the diastolic filling the greater the initial length of the muscle fibres produced by stretching, and the greater the initial length of the muscle fibres the greater the force of their contraction. If the pressure in the aorta is unusually high the pressure in the left ventricle must rise to an unusual height in order to open the semilunar valves. To raise the intraventricular pressure the muscle must develop a greater isometric tension and it can only accomplish this, as has been pointed out, by increasing the length of the muscle fibres. When the pressure in the aorta is suddenly raised, the left ventricle for a few beats throws out less blood than usual during systole. As a result the diastolic volume increases with each beat as the ventricle fails to expel all the blood it re-

ceives, and thereby the muscle fibres are lengthened more and more and their isometric tension increased. (Fig. 12.) The systole now becomes more powerful and the normal amount of blood is expelled against increased resistance in the aorta. The stretching of the heart muscle in this physiological dilatation has enabled the heart to do more work and to compensate for the rise of blood-pressure in the aorta (Starling).

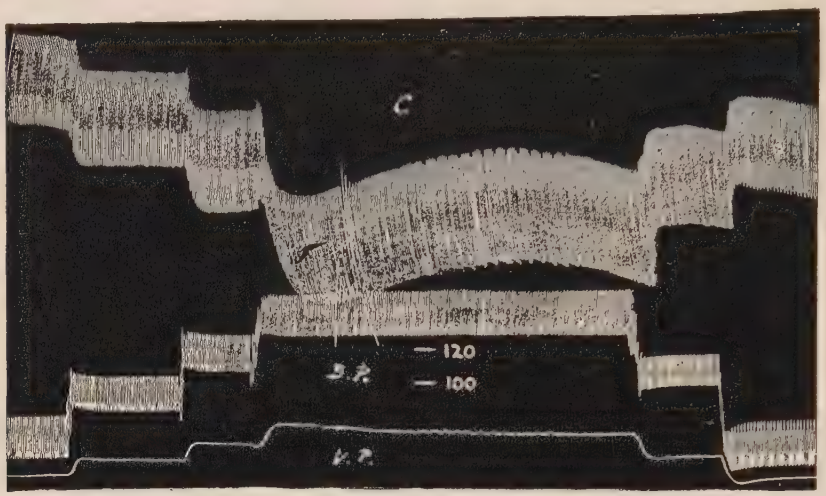


FIG. 12.—Effect of raising and lowering the arterial resistance on the volume and output of the ventricles in the heart-lung preparation. C. the cardiometer tracing. The top of the first line to the left was the position of the lever at the end of the first systole recorded in the experiment. Down strokes represent increase of volume. B.P., arterial pressure; V.P. venous pressure. A rise of arterial resistance causes an increase of the diastolic volume and the systolic residue. (Patterson, Piper and Starling, J. Physiol., XLVIII.)

The Cardiac Cycle.—The auricles have weak, thin walls and hence the work demanded of them must be slight. With the opening of the auriculoventricular valves the blood flows readily into the dilating ventricles. The ventricular filling takes place rapidly during the first part of diastole, while the amount of blood added by the auricular contraction is slight. If during the passage of blood into the ventricle the pressure rises unduly and the resistance to the onward movement of the blood becomes too great for the auricle to overcome, the blood can readily flow back into the pulmonary veins as they have no valves to check this flow. This is evidently a safety mechanism that nature has provided. The tenia terminalis, a muscle band, lying obliquely along the junction of the superior vena cava and the right auricle, aids in preventing the reflux of blood into this vein during contraction of the auricle. With the onset of the ventricular systole the auriculoventricular valves close and the

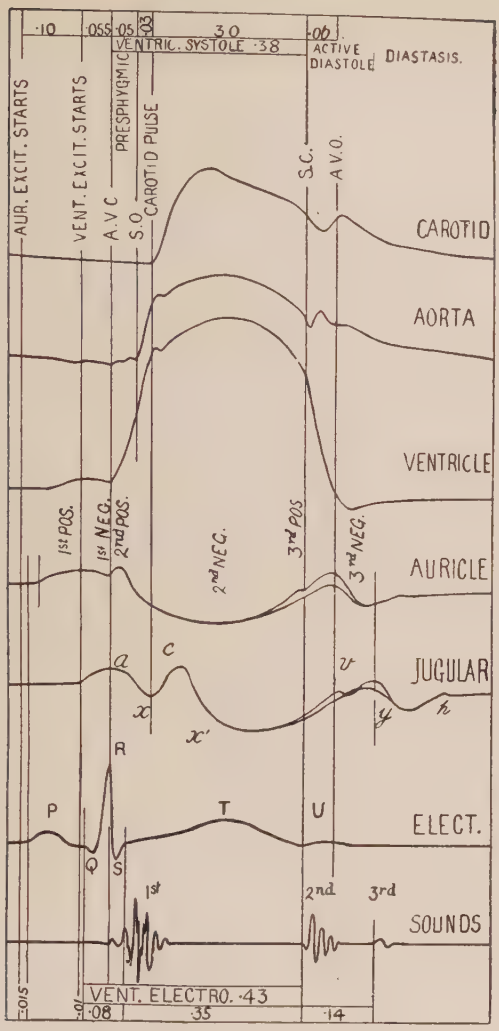


FIG. 13.—"A diagram intended to represent the curves of pressure in the carotid, aorta, left ventricle and right auricle of the human subject; showing the supposed or ascertained time relations of these curves, the jugular pulse curve, the electrocardiogram and apical heart sounds to each other. The diagram has been constructed so far as possible from curves taken from man, but where such are non-existent the diagram has been completed by curves obtained from the dog. . . . The times at which the excitation wave begins in auricle and ventricle are represented by vertical lines; the movements of the valves are also represented in a similar way. The scale of abscissae is 1 mm. = 0.015 sec. A.V.C. and A.V.O. = closure and opening of auriculoventricular valves, respectively; S.O. and S.C. = opening and closure of semilunar valves respectively. Other verticals are drawn at convenient points and the chief time intervals are marked above and below in seconds." (Lewis, "The Mechanism and Graphic Registration of the Heart Beat," Shaw and Sons.)

tension of the ventricular muscles rapidly increases but the muscle fibres do not shorten. This is an isometric contraction. The pressure rises rapidly within the closed ventricular chambers. The period that elapses between the onset of the ventricular systole and the opening of the semilunar valves is known as the isometric period. It was formerly called the presphygmic period. It lasts 0.06 to 0.07 of a second. When the increasing pressure in the ventricle exceeds that in the aorta and the pulmonary artery the semilunar valves fly open. Then the muscle fibres begin to shorten (isotonic contraction) and the contents of the ventricle are discharged. The pressure first continues to rise after the opening of the valves, and then begins to fall. A considerable part of the ventricular output is expelled while the pressure is falling. When the pressure drops below that in the artery the semilunar valves quickly close and the systole is ended. (Fig. 16.)

The Work of the Heart.—The human heart usually expels with each systole between 60 and 80 cc. of blood against a high pressure within the aorta. This pressure usually amounts to 120 mm. or more of mercury. It does this amount of work even when the body is at rest about 70 times in the minute. The heart also imparts velocity to the blood but as this represents only 1 per cent of the work of the heart it may be disregarded. The work of the heart in a single contraction can be increased in two ways. (1) By raising the pressure within the aorta. This will increase the amount of work required to open the valves and to expel the blood. (2) By increasing the flow of blood from the auricles to the ventricles during diastole. This requires the ventricle to expel a greater quantity of blood during systole against an unreduced aortic pressure.

There is no satisfactory method for measuring the output of the human heart and hence the work done by the heart in health and disease cannot be directly determined. Indirect methods involving the use of special laboratory methods have given varying results. The published figures for the systolic output per beat during rest vary from 26 cc. (Krogh and Lindhard) to 125 cc. (Douglas and Haldane).

The Effect of Exercise on the Heart's Work.—Active muscles require a greatly increased supply of blood. The mean blood-pressure during exercise rises sometimes 50 per cent or more. The systolic output per beat is probably increased and the heart-rate increases so that the minute volume is much greater. Hence the work of the heart, which, as stated, is the product of the minute-volume by the resistance, is enormously increased. After the exercise ends the blood-pressure, if the heart be healthy, quickly drops to normal, but the fall of the pulse-rate is more

gradual. Exercise is the great test of cardiac efficiency. When the heart is weak the rapid pulse-rate after exercise usually persists for a longer time after exercise, and the behavior of blood-pressure may vary from that in health. Methods of determining cardiac insufficiency by functional tests involving changes in pulse-rate and blood-pressure have proved untrustworthy. The effect of exercise on the patient's sensations, —breathlessness and palpitation, must never be neglected in determining the existence and the degree of cardiac weakness.

The Nerves of the Heart.—Both vagi send branches to the heart.

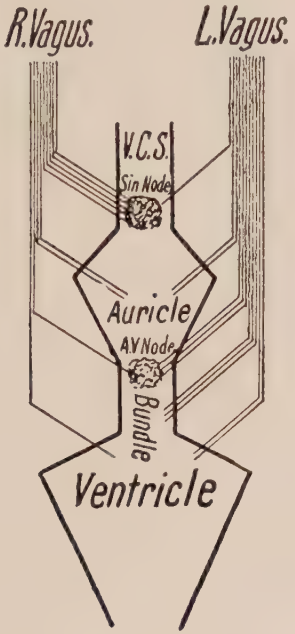


Fig. 14.—Distribution of the ends of the vagus in the heart. (After Cohn. Romberg, "Krankheiten des Herzens und der Blutgefässe," Ferdinand Enke.)

The right vagus supplies chiefly the sinus node and the left vagus the auriculoventricular node, His's bundle, and the ventricular part of the conducting system. (Fig. 14.) The vagus is the inhibitory nerve of the heart. It slows the rate and lessens the irritability and conductivity. The accelerators increase the heart rate and the irritability and conductivity of the muscle. The right accelerans acts on the sinus node and the tertiary centers in the right ventricle. The left accelerans influences the atrioventricular node and the tertiary centers in the left ventricle. Both vagi and accelerators are normally in tonic activity. An efferent nerve, the depressor, is also connected with the heart. Impulses passing from the

heart to the brain along this nerve cause a fall of the general blood-pressure due to action on the vasomotor center in the medulla.

Reserve Power of the Heart.—The ability of the healthy heart to respond immediately to increased demands made upon it is due to what is called its reserve power or range of accommodation. The heart is the most efficient motor in existence and one of its remarkable features is this ability to adapt itself to the varying conditions of everyday life. A simple illustration may make this clear. A boy lying on a couch dozing is suddenly awakened by the sound of a passing fire engine. He jumps to his feet and goes at top speed down the street. He does this without difficulty and if accustomed as most boys are to vigorous exercise he can run quite a distance without any difficulty in breathing. The increase in the work done by the heart when the boy is running over that when resting quietly is enormous. It has been estimated that when a heart is pushed to its maximum effort it does thirteen times as much work as when a person is at rest. The reserve power is probably due to the increased contractile power of the muscle fibres of the heart when stretched.

Law of the Heart.—Increased filling of the ventricles with blood during diastole lengthens the muscle fibres. Within wide limits lengthening of the heart muscle fibres increases the work they can do in contracting because the initial tension rises when the muscle fibres are stretched (Frank). Hence, dilatation of the normal heart with blood lengthens the muscle fibres and thus enables them to do more work. If the aortic pressure is suddenly raised the left ventricle may be unable to empty itself completely during the next systole. The residue of blood together with the amount flowing into the ventricle from the auricle will increase the size of the ventricular chamber. This dilatation by stretching the muscle fibres will enable them to contract with greater force and thus empty the ventricle more completely.

Cardiac Insufficiency.—Continued effort will result in exhaustion of the reserve power. If a boy keeps on running he will after a time be conscious of difficulty of breathing and pounding of the heart. If he does not then stop the respiratory distress will soon become so great that it will compel him to rest. The labored breathing is evidence that he has used up his cardiac reserve. (Fig. 15.)

If the heart is weak less effort is required to produce breathlessness than if the heart is strong because the reserve power of the weak heart is less. Often in advanced disease of the heart muscle the reserve power is so completely exhausted that the patient is breathless when at rest in bed.

If the heart muscle is unable to do its work, that is to supply the muscles and other parts of the body adequately with blood, the condition is called cardiac insufficiency or heart failure. This is due to reduction of the reserve power of the heart.

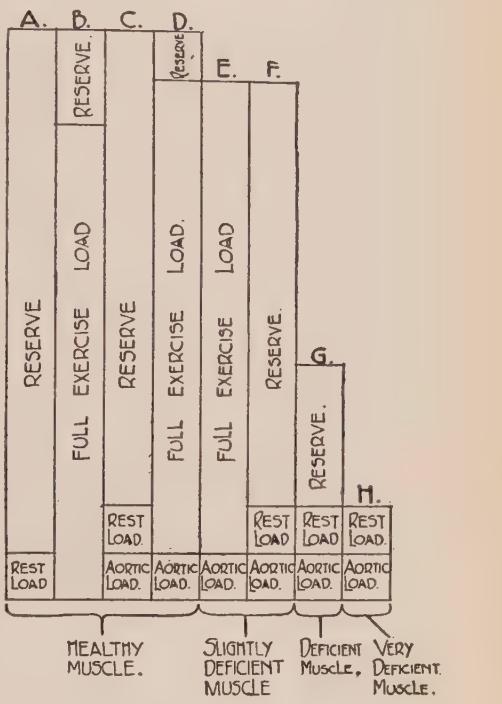


FIG. 15.—"The accompanying diagram expresses, so far as I am able to express it diagrammatically my conception of heart failure in relation to the load of work which the heart has to bear. I represent the capacity of the normal heart for work in the first column (A) of the diagram, dividing this column into two parts, to represent the load upon the heart while the body is at complete rest (rest load) and the reserve. The capacity for work in reserve I have intentionally drawn many times greater than the work of the heart muscle in conditions of rest. In the second column (B) I represent the heart fully loaded by exercise, putting forth the maximal quantity of blood against an increased resistance. I represent this full exercise load as many times greater than the rest load, but still show a reserve, though this is necessarily much diminished. In the third column (C) is represented a hypothetical instance of aortic reflux in which the muscle of the heart is supposed to be perfectly healthy, and I subdivide the whole column into the rest load, the aortic load and the reserve. My objects in introducing this column are to represent the aortic load as no greater than the rest load and to show the reserve as little diminished.

"The reserve is still capable of absorbing the full exercise load without being itself completely exhausted (a condition represented in column D.) Column (D) represents an ideal case, to which, however, certain instances of aortic reflux in soldiers closely approach, for the patients to whom I refer present an almost perfect exercise tolerance. Columns (E) and (F) represent these patients more truly, for in such aortic cases there is probably always some deficiency of the muscle; I represent the reserve as just exhausted by the full exercise load column (E), but as ample for all the usual calls of every day life column (F). In column (G) I represent the heart muscle as definitely deficient, the rest load and aortic load together occupying a considerable proportion of the whole column, and the reserve as greatly diminished as compared to the normal. Such a heart is incapable of meeting anything but small demands for increased work. Lastly, in column (H) a heart is represented in which the muscle is very deficient and in which the rest load and aortic load are all that the heart is capable of carrying. Such a heart is just capable of maintaining the circulation when the body is at complete rest, but it is capable of no more.

The whole diagram stresses the view that the heart fails because its reserve is exhausted by damage of its muscle. (Lewis, "The Soldier's Heart and the Effort Syndrome," Shaw and Sons.)

The Filling of the Aorta.—With each systole the left ventricle suddenly throws a certain quantity of blood into the aorta. This output of blood is due to the rise of pressure in the ventricle, produced by the contraction of its muscular wall, to a point above that in the aorta. (Fig. 16.)

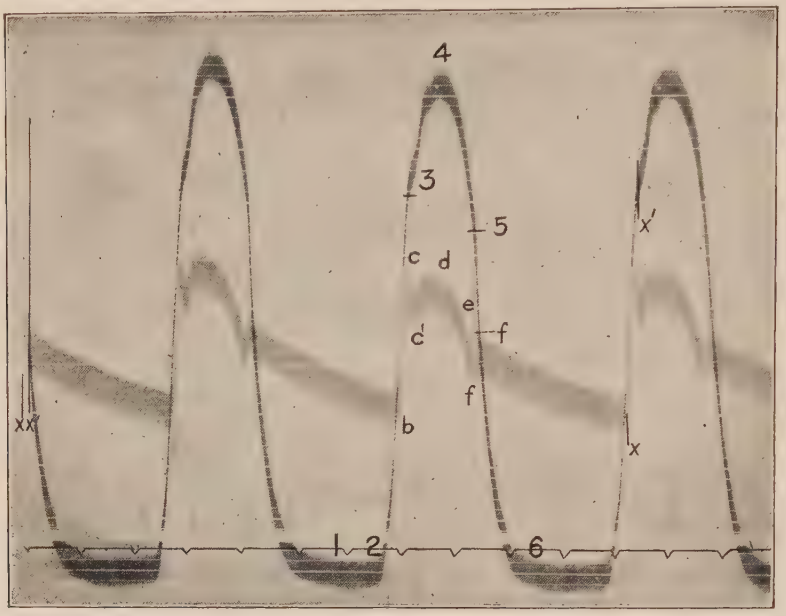


Fig. 16.—Simultaneous intraventricular and subclavian pressure curves. x–x', relative positions; T, time in ⅕ seconds; 1–2, auricular rise; 2–3, isometric period; 3, opening of semilunar valves; 3–4, ascending limb of ejection period; 4–5, descending limb of ejection phase; 5–f, incisura; f–6, isometric relaxation, b–c, primary oscillation; d, systolic summit; e–f, incisura. (Wiggers, "Circulation in Health and Disease," Lea and Febiger.)

The average amount discharged is about 60 cc. The aorta is distended with blood and the pressure in it is high when the aortic valves open, but the pressure is increased when this additional blood is thrown into it. This new blood might be accommodated in two ways. First, it might push onward the column of blood in the aorta and thus make a place for itself. This method would be the only one possible if the aorta was a rigid tube as the blood is an incompressible mass. As a result as much blood would be discharged into the capillaries with each systole as entered the aorta from the ventricle. To move forward the whole column of blood sufficiently to accommodate the additional blood would require much greater force than is exerted by the ventricle. Secondly, the systolic output leaving the ventricle under high pressure might distend the walls of the first portion of the aorta as its walls are elastic and distensible. This

is actually what occurs. Much less work is required by this method for providing room in the aorta for the new blood than by the first described. True a small part of the energy, only about one per cent, is expended in forcing forward a little the blood column, in other words by increasing the blood flow. The balance—99 per cent—is stored as potential energy by distending the walls of the aorta.

The Pulse Wave.—The rise of pressure in the aorta during the systole of the heart is transmitted from the aorta to the peripheral arteries in the form of a wave. This is called the pulse. The pulse wave travels rapidly, in fact from six to ten meters per second. The speed of this wave of increased pressure is so swift that it reaches the smaller arteries before the cardiac systole is ended. It takes about a tenth of a second to travel from the aorta to the radial artery. The velocity of the blood flow is much less than that of the pulse wave. It probably averages about three-tenths of a meter per second. Wiggers [1] gives an apt illustration of the relation of the pulse wave to the blood flow. He compares the pulse wave "to an impact transmitted through a train of cars in motion, the rate at which the impact travels is entirely independent of the speed of the train and may in fact occur in an opposite direction."

The Curve of Aortic Pressure.—The pressure in the aorta reaches its maximum shortly after the semilunar valves open. This is called the systolic pressure. It remains high for a time, the pressure curve forming a plateau (see Fig. 13). It then falls gradually as does the pressure in the ventricle with which it forms one cavity while the aortic valves remain open. The fall is soon checked by the closure of the semilunar valves. During the remainder of diastole the fall is gradual and continuous. The minimum pressure is termed the diastolic pressure. The difference between the systolic and the diastolic pressure is called the pulse pressure.

The Blood Flow.—When the semilunar valves close at the end of systole the elastic walls of the aorta distended by the blood thrown in from the left ventricle contract. The energy liberated by the contraction of the aorta forces the blood along the arteries. This force continues to act throughout diastole. As a result the flow of blood in the arteries is continuous throughout the cardiac cycle. Were the walls of the arteries rigid the onward flow would be intermittent, occurring only in systole.

The Cause of the Pulse.—The arterial pulse is usually felt in the radial artery at the wrist. This is owing to its accessibility and also because it is more easily palpated than any other artery of goodly size.

[1] "Modern Aspects of the Circulation in Health and Disease," 1915, 101.

The pulse that one feels in the artery following each systole of the heart is due to the onward passage of the pulse wave from the aorta. It is the sudden rise in pressure within the artery pressing against the finger that is felt and not the onward flow of blood. It is important to emphasize the cause of the pulse, because the old error still lives in the minds of many practitioners that the pulse is due to the expansion of the artery. The visible movement seen in health in the carotids and sometimes in the radials is due not to expansion and contraction of these vessels, but to a displacement of the entire artery. During the ventricular systole a straight vessel like the carotid can be seen to be slightly tightened like a cord and during diastole to be relaxed. (Mackenzie.)

A finger placed on an artery does not detect a pulsation unless the artery is pressed against a bone. The radial artery is more or less flattened against the radius by the finger in the interval between the beats. The next pulse, that is the next rise in pressure, attempts to restore the cylindrical form of the vessel and it is this change in shape of the artery that is mistaken for an actual expansion. Surgeons know, as Lister pointed out, that arteries in soft parts pulsate so little that they cannot be distinguished from the other structures by the expansion of their walls unless pressed against some unyielding structure. They are easily mistaken for nerves or tendons unless accidentally cut. Mackenzie has demonstrated by pulse tracings that the sphygmogram represents a change of pressure and not an expansion of an artery. Fig. 17 gives a tracing

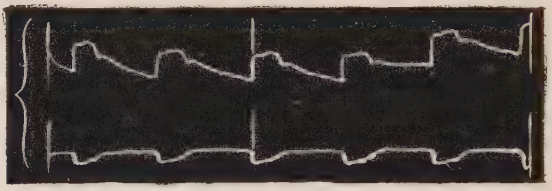


Fig. 17.—The upper tracing was taken with the receiver over the carotid artery, at the same time as the lower one was taken with the receiver placed by the side of the carotid artery. The lower tracing is the inverse of the upper. (Mackenzie, "The Study of the Pulse," Young J. Pentland.)

made with the receiver directly over the carotid artery, and a second tracing obtained with a receiving cup beside the carotid artery. The latter is seen to have a systolic fall, while the carotid tracing shows a systolic rise. The tracing made at one side of the carotid is what Mackenzie terms an "inverted sphygmogram." It is due to a displacement of the carotid as a whole away from the receiver during the cardiac systole. If the pulse wave shown in the tracings were due to an expansion of the

vessel it would have the same shape in a sphygmogram made beside the carotid as over that vessel, because if the artery expanded it would thrust out all the tissues surrounding it.

The pulse wave becomes less in the smaller arteries and arterioles due to increasing resistance, and is not seen usually in the capillaries and veins. The smaller arteries have walls richer in muscle tissue than the larger vessels and poorer in elastic tissue. The muscular tissue is under control of the vasomotor nervous system which regulates the diameter of the vessels. When an organ, or part of the body, is working, the artery that supplies it is dilated, while the arteries that lead to the structures that are resting are contracted. In this way, through the action of the vasomotor nerves, parts that need blood are supplied with it, without necessarily throwing more work upon the heart. The control of the nervous regulation of the size of the blood-vessels is located in the vasomotor center in the medulla oblongata. How great a rôle is played by peripheral factors either of a nervous or chemical nature remains unknown.

Since the nerves maintain at all times a considerable constriction (tonus) of the vessels the peripheral resistance to the onward flow of blood is high. The greater the resistance in the smaller arteries and capillaries the higher will be the blood-pressure.

An artery like the radial differs much in size from time to time according to the degree of contraction of the muscle in its walls. It is also believed that actual hypertrophy of arterial muscle may account for increase in ease of palpation.

HYPERTROPHY OF THE HEART

If a healthy voluntary muscle is made to do increased work for a considerable time, it usually increases in size. The individual muscle fibres in an hypertrophied muscle are thicker than normal. With increase in size is a gain in strength so that the hypertrophied muscle can do more work than the normal muscle. Growing muscles always hypertrophy when exercised, but it is not true that increased work always causes an adult muscle to grow larger; and, further, muscles which are the seat of various pathological processes do not hypertrophy (Hasebroek).

The involuntary muscles of the stomach, intestine and bladder, hypertrophy above a chronic obstruction owing to the increased work thrown upon them.

The size of the heart is proportional to the size of the body. A large

man has normally a much larger heart than a small man. There is a more direct relation between the weight of the heart muscle and the mass of skeletal muscles than to the whole body weight. Fat tissue bears no relation to the size of the heart. As fat may make up a considerable but variable part of the weight of the body, the amount of fat is a disturbing factor in comparing the weight of the heart to that of the body, and hence an allowance should be made for fat, bones and other inactive tissues. By hypertrophy of the heart is understood an increase in weight that is disproportionate to the mass of skeletal muscles. The average normal weight of the heart in man is usually given as 300 grams and in a woman 250 grams. But these figures have only a relative value, and must always be considered in connection with the body weight. The normal weight of the heart of a man weighing 40 to 50 kilograms would be 230 grams, while 376 grams would be normal for one weighing 95 kilograms.

Hypertrophy of the heart is common, but its origin is often obscure. One form can be readily produced experimentally in animals by tearing the aortic valve. When the aortic valve is rendered incompetent by injuring the cusps the left ventricle will do more work because at each systole it will have to expel the blood that regurgitates from the aorta during diastole as well as that received from the left auricle. Definite hypertrophy of the left ventricle has been found to occur in two and a half weeks after the production of this valvular lesion (Tangl). In a month the heart of a rabbit in which two aortic cusps had been perforated was about three times the weight of the heart of a control animal (Gelbart).

When aortic insufficiency is experimentally produced by tearing the valve, the left ventricle at once dilates during diastole and thereby is able to hold the usual amount that flows from the auricle in addition to that which regurgitates from the aorta. This is called a compensatory or tonogenic dilatation. It is probable that hypertrophy of the heart occurs only as a result of such a tonogenic dilatation. The dilatation by lengthening the muscle fibres increases the initial tension. According to this view it is the increase in initial tension long continued that produces hypertrophy.

When the hypertrophied heart muscle overcomes the effect of a valvular lesion, the condition is called a compensatory hypertrophy. The extent of the hypertrophy depends not only upon the valvular lesion, but also on the demands made on the heart by bodily exertion. For example, aortic insufficiency developing in a longshoreman would be expected to lead to much greater hypertrophy than in a cripple confined in

his bed as a result of some other disease such as arthritis deformans or phthisis.

Little is known regarding the so-called work hypertrophy of the heart. Reasoning from analogy one would expect that great physical exertion if long continued would lead to increase of the heart muscle. It is true that animals that are very active have hearts that are relatively large compared to the weight of the body. The ratio of the weight of the heart to the body weight is about five times as great in birds as in man. It holds in some instances, at least, for those of the same species, that birds that are most active have the largest hearts. Greyhounds and race horses are, also, cited as examples, and the wild rabbit has a larger heart than the rabbit kept in a pen. There is some doubt, however, whether in these animals the ratio of the heart weight to the total weight of the skeletal muscles is increased. Two puppies of the same litter were studied by Külbs. One was made to work a treadmill daily and the other was given little exercise. When killed the heart of the more active dog was not only heavier than the control, but the ratio of the heart weight to the weight of the muscles was increased.

The clinical evidence of work hypertrophy is unsatisfactory. The heart of ski runners in Denmark is said to be frequently hypertrophied. The size of the heart of recruits in the army increases as a result of military drill, according to Schieffer who made observations with the orthodiagraph. This radioscopic apparatus gives a silhouette which is the actual size of the heart. This method, however, would indicate dilatation of the heart rather than hypertrophy, as hypertrophy alone would not give a sufficient increase in the size of the cardiac shadow to be recognized in the orthodiagram. Probably, however, hypertrophy was associated with the dilatation. Increase of the skeletal muscles doubtless occurred, and possibly the ratio of heart weight to muscle weight was not increased. If so, true cardiac hypertrophy, according to the accepted definition, did not result. Besides, studies made during the war failed to show that arduous campaigning even in mountain regions led to cardiac hypertrophy.

This problem of cardiac hypertrophy can be studied more satisfactorily at the autopsy table than during life as there is no adequate clinical method for determining the degree of hypertrophy. A recent pathological investigation of a large series of cases in which the left ventricle was enlarged failed to show any that could be regarded as a work hypertrophy (Hecht).

Nearly fifty per cent of all persons over sixty years of age have hyper-

trophied hearts (Councilman). This is not a physiological process nor is it definitely the result of arteriosclerotic changes. Practically all aged persons have arteriosclerosis of the aorta and its branches and it may be as advanced in a subject with a normal heart as in one with a heart that is hypertrophied. The weak heart of old age, known clinically as the senile heart, is commonly an hypertrophied heart. The cause of the hypertrophy is unknown, although it is probably associated with some primary alteration in the heart muscle that is not discoverable on microscopic examination. Some of the cases are possibly due to sclerosis of the coronary arteries.

Up to the age of forty it is very rare to find a person who has an hypertrophied heart without assignable cause. Above the age of fifty it is not uncommon to find a definitely hypertrophied heart without evidence of disease elsewhere. A kidney lesion involving the vessels of the glomeruli is the most common apparent cause of cardiac hypertrophy. This statement is based on the fact that an arteriosclerotic contracted kidney is found in about 50 per cent of the cases of hypertrophy of the left ventricle. But there is a tendency to overemphasize the glomerular lesion. The fact should not be overlooked that the amyloid kidney with its extensive glomerular disease does not cause cardiac hypertrophy. Valvular lesions are responsible for approximately 25 per cent of the cases of cardiac hypertrophy (Hecht).

It is held that hypertrophy is limited to the portion of the heart that is called upon to do extra work. Disease of the valves may lead to narrowing of the orifice (stenosis) or to imperfect closure of the cusps (insufficiency). If the aortic orifice is obstructed more work is required of the left ventricle to expel its blood into the aorta. This regularly leads to hypertrophy of the wall of the left ventricle, but to less marked hypertrophy than does aortic insufficiency.

In aortic insufficiency there is a regurgitation of blood from the aorta into the ventricle during diastole through the leaky valve. The ventricle, if it is to empty itself, must discharge this extra amount of blood together with the normal amount received from the left auricle. Hence during systole more work is required of the left ventricle and it undergoes hypertrophy.

In mitral insufficiency the left ventricle returns part of its contents, during systole, through the mitral orifice into the left auricle, and forces part into the aorta. But, to maintain the blood supply of the organs, it must continue to throw the normal amount of blood into the aorta. This additional work put upon the left ventricle causes it to undergo hypertrophy as one would expect.

Pure mitral stenosis puts no extra work on the left ventricle and hypertrophy of this portion of the heart does not occur. The part of the heart that becomes hypertrophied in obstructive lesions of the valves is above the diseased valve, using the term "above" with reference to the direction of the blood stream. In mitral stenosis and to a less extent in mitral insufficiency increased work is thrown on the left auricle. The task is too great for the weak thin wall of the auricle and as a result, the blood is dammed back in the lungs. The pulmonary veins are capable of holding a great amount of blood by distention of their walls. In this way a reservoir is provided for the left auricle and for a time no rise in the pressure in the pulmonary veins occurs. Finally, if the condition persists, sufficient blood accumulates behind the left auricle to raise the pressure in the pulmonary veins. In animals with experimental mitral lesions no increase of pressure in the pulmonary artery occurs after a rise in the veins. Straub concluded from this that the hypertrophy of the right ventricle, which undoubtedly occurs in patients with mitral stenosis and insufficiency, could not be a work hypertrophy. Gerhardt showed that the failure of the blood-pressure to rise in the pulmonary artery of animals was due to an inadequate blood supply to the right heart. When the volume of blood flowing to the right ventricle was increased in animals with mitral disease the blood-pressure in the pulmonary artery rose. In mitral disease as seen in patients with its slow development the volume of blood in the general circulation is sufficient to raise the blood-pressure in the pulmonary artery (Gerhardt). The right ventricle has to contract with greater force to propel the blood through the lungs when the pressure is raised in the pulmonary artery. In consequence the right ventricle hypertrophies in mitral stenosis.

The hypertrophic hearts are weaker than normal hearts in the sense that their reserve power is relatively reduced. This fact does not imply that a heart that has undergone enlargement of its muscles is weaker than that particular heart would have been if it had not undergone hypertrophy.

Combined lesions are common. Mitral stenosis is usually associated with mitral insufficiency. When this occurs the obstruction and the insufficiency lead to hypertrophy of the right ventricle and the insufficiency to hypertrophy of the left ventricle.

Kyphoscoliosis interferes with the circulation of blood through the lungs. If of high degree it leads to hypertrophy of the right ventricle because more work is required to force the blood from the right side of the heart to the left ventricle. Emphysema diminishes the number of

the capillaries and throws more work on the right ventricle which explains its hypertrophy in this disease.

If extensive adhesions bind the heart to the pericardium and also to the mediastinum and chest wall, the work of the heart will be increased at each contraction by the resistance of the structures to which it is attached, and hypertrophy of either one or both ventricles will result. In cases with adhesions limited to the two layers of the pericardium hypertrophy results if the adhesions are extensive. The normal attachments of the parietal pericardium posteriorly to the mediastinal tissues, laterally to the hiluses of the lung, and anteriorly to the sternum are sufficient to account for hypertrophy. (Karsner.)

DILATATION OF THE HEART

Pure hypertrophy of the heart is of rare occurrence, and it does not produce sufficient enlargement of the heart to be recognized by physical examination. Increase in size of the heart is the result of dilatation of the cardiac chambers, a condition in which the heart contains a much larger amount of blood than normal at the end of systole. One reads in the older textbooks on pathology of a concentric hypertrophy, that is an hypertrophy without dilatation of the cavities, but it is doubtful if this ever occurs. In all the cases of hypertrophy of the heart that the student will study in the clinic, the hypertrophy will be combined with dilatation. An increase in the unit volume of blood received in diastole and expelled during systole does not cause a sufficient increase in the size of the heart to be recognized clinically (Straub). In violent exercise the systolic output of blood is greatly augmented, and more blood enters the ventricle during each diastole than occurs in any form of valvular heart disease, no matter how severe the valvular defect may be. Yet in exercise the size of the heart determined by the orthodiagraph is not increased and hence the dilatation in valvular disease is never due to increased intake and output of blood. As a matter of fact in valvular lesions including aortic insufficiency the systolic output is not greatly increased.

Normally the ventricle does not completely empty itself during systole. There remains a systolic residue and the amount of blood at the end of diastole consists of the blood that has entered the ventricle from the auricle plus this residual blood. If Valsalva's experiment,—violent expiratory effort with glottis closed,—is made by a person whose heart is being observed radioscopically, the heart will be seen with each beat to diminish in size for a number of contractions. In this experiment the flow of

venous blood to the heart is lessened and the ventricles empty themselves more and more completely, and thus reduce the rest volume.

There are two forms of dilatation of the heart: (1) the compensatory or tonogenic and (2) the congestive or myogenic (Moritz). The increase of the rest-volume, that is the residue of blood left in the heart at the end of systole, is one of the factors of safety in the animal mechanism. It furnishes a means of overcoming increased resistance to the discharge of blood from the ventricle into the aorta. If the residue of blood left in the heart at the end of a systole is increased, but the condition of the circulation is otherwise unchanged, then, during the following diastole, the volume of the ventricle will be larger than during the preceding diastole, because it will receive the usual amount of blood from the auricle, and this, plus the larger systolic residue, must increase the volume of the ventricle. As a result of the increased filling of the ventricle the initial isometric tension of the muscle fibres will be increased and this will enable them to contract with greater force and thus raise the intraventricular pressure to a higher point than during the previous systole. The increase in intraventricular pressure developed during systole will overcome the increase of pressure in the aorta. In this way the dilatation of the heart will have a beneficial effect because it enables the heart to overcome increased resistance.

In *aortic stenosis* compensatory dilatation always occurs If this condition is produced experimentally and the changes in the volume of the ventricles studied it will be found that during the systole immediately following the production of the lesion the volume of blood expelled from the heart into the aorta is diminished, and this of course causes an increase in the volume of the ventricle at the end of systole as the residue volume of blood is increased. With each contraction the dilatation of the heart increases until the amount of blood expelled during systole equals the previous systolic output. (Fig. 18.) The dilatation, which is then considerable, remains after this unchanged, but the ventricle now throws out at each systole as much blood as it receives during diastole. The dynamic consequences of increased pressure in the aorta such as occur whenever the general blood-pressure is elevated is similar to that in aortic stenosis.

In *aortic insufficiency* the volume of the ventricle is increased at the end of diastole as the ventricle contains the amount of blood that regurgitates from the aorta during diastole plus the systolic residue and the blood received from the auricle. If the contractile power of the ventricle is not impaired it will simply expel more blood during the next systole

and the systolic residue will not be increased. Hence dilatation of the left ventricle does not belong to the primary picture in aortic insufficiency. The dynamic effect of the regurgitation of blood into the left ventricle is a dilatation sufficient to compensate for the volume of blood that re-

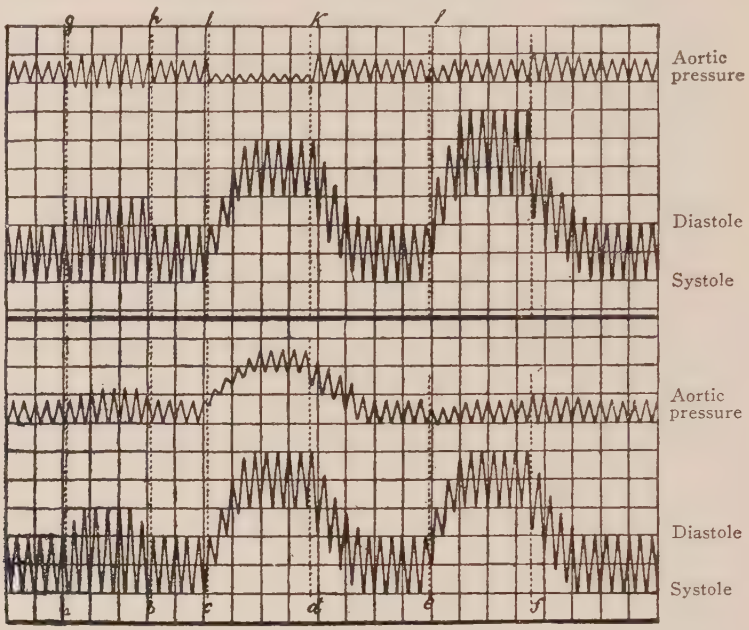


FIG. 18.—Curves of aortic pressure and ventricular volume. *g–h*, aortic insufficiency; *l–k* aortic stenosis; *l–*, mitral insufficiency; *a–b*, increased volume of output (Schlag volume) *c–d*, increased resistance; *e–f*, injury to contractile power of heart; *f–* increase in contractile power of heart. (Straub, Deutsche med. Wchnschr., XLV.)

gurgitates and an increase in the systolic output. Cases of aortic insufficiency with hypertrophy of the left ventricle when studied at the autopsy are always associated with dilatation of the left ventricle.

In *mitral insufficiency* according to the recent work of Wiggers and Feil there is very little regurgitation of blood from the left ventricle to the auricle. They demonstrated that this was true even when no valves exist between the two chambers provided the increase in intraventricular pressure occurs rapidly. If this work is confirmed, it is evident that the compensatory dilatation of the heart would be slight. Straub has maintained that a considerable compensatory dilatation occurs in mitral insufficiency. His experiments indicated that the back flow of blood through the mitral orifice produced a steady loss of ventricular pressure in both the isometric and ejection periods. This loss of pressure could

only be overcome by a great increase of the initial tension. To obtain this requisite initial tension a marked compensatory dilatation would be necessary. The optical tracing of Wiggers and Feil reproduced in Fig. 19, shows clearly that even when gross mitral insufficiency exists the leak

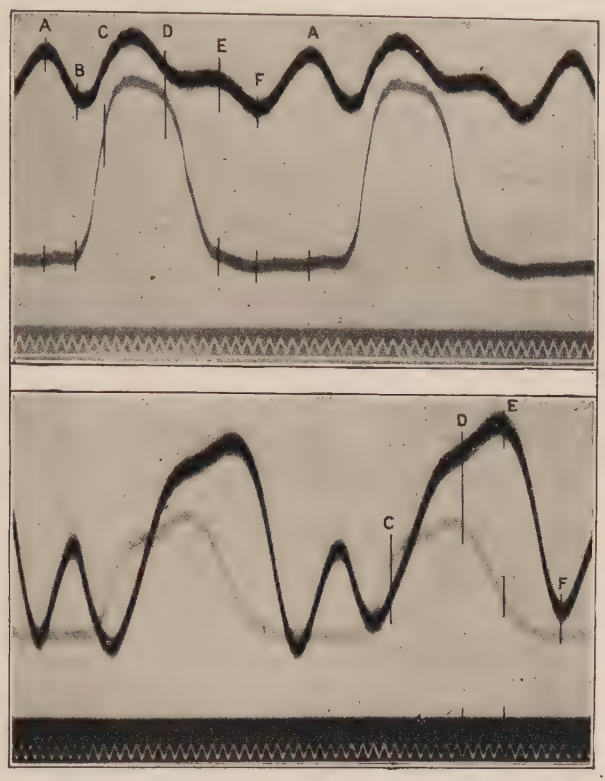


FIG. 19.—Two segments, showing auricular volume curves (dark curve) and intraventricular pressure curves (lighter curve). Upper segment, normal curves; lower segment, during mitral regurgitation. *C*, beginning of systolic ejection from the ventricle; *D*, end of ventricular systole; *E*, mitral valve opens and diastolic inflow into ventricle begins. The curves show that during the isometric contraction period which ends at *C* the auricular volume increases but little more when the mitral valves are incompetent than when they are normal. (After Wiggers and Feil. Wiggers, "Modern Aspects of the Circulation in Health and Disease," second edition. Lea and Febiger.)

has very little effect on the auricular volume during the isometric period. Hence Straub's conclusions cannot be accepted.

If the contractile power of the heart is suddenly injured, the ventricles empty themselves less completely than before until the post systolic residue has led to such dilatation of the heart that the increased initial tension of the injured muscle enables it to overcome the normal resistance in the aorta and to furnish the arteries with the requisite amount of blood at

each systole. This myogenic or congestive dilatation differs only from the simple compensatory form in the relation of the strength of the ventricular muscle to the resistance to be overcome. In the simple compensatory form the resistance is increased while the strength of the ventricular muscle is normal. In the myogenic type of dilatation the resistance to be overcome is normal, but the strength of ventricular muscle is impaired.

In *mitral stenosis* there is a compensatory dilatation of the right ventricle. The obstruction at the mitral orifice first leads to compensatory dilatation of the left auricle. The pressure in the auricle is raised when the auricular muscle by more forcible contraction forces the blood through the narrowed initial orifice. At the same time, the pressure in the pulmonary veins is increased as no valves separate them from the auricle. Increase of pressure in the pulmonary veins is followed by increased pressure in the pulmonary artery. To expel the normal amount of blood from the right ventricle into the pulmonary artery against increased resistance requires the same adjustment of the dynamics that occurs in aortic stenosis, described above. The right ventricle when the pressure in the pulmonary artery is suddenly raised expels at first less blood in systole. The postsystolic residue of blood increases until the dilatation thus produced stretches the muscle fibres of the right ventricle to the point where the increased tension enables the ventricle to discharge the normal amount of blood into the pulmonary artery in spite of the increased resistance in that vessel.

In all the conditions described the dilatation of the heart if persistent for several weeks regularly leads to hypertrophy of the walls of the dilated chamber. It is very rare indeed at autopsy to find dilatation of a portion of the heart unassociated with hypertrophy. Acute dilatation of a healthy heart to an extent that can be recognized clinically has never been observed. It occurs occasionally in cases of paroxysmal rapid heart action, but hearts in which these attacks occur cannot be regarded as quite normal. It probably never occurs as a result of extreme physical exertion if the heart is healthy. An acute dilatation of a heart with a diseased myocardium may take place, but it is certainly not common.

If a heart has once become so dilated that its enlargement can be definitely recognized it usually remains dilated throughout life. This means in terms of dynamics that the systolic residue of blood if it once becomes greatly increased in amount very rarely returns to normal limits, and usually persists with little diminution.

The size of the heart is no indication of its functional condition. A large heart may be so efficient that laborious work may be done without

conscious effort, or so weak that it cannot maintain the circulation with the body in rest. Enlargement of a heart, determined by physical means, is a measure of its dilatation, but it is equally true that the degree of dilatation of a heart is no index of its strength or weakness.

CHAPTER II

PROCEDURES IN PHYSICAL DIAGNOSIS AS APPLIED TO DISEASES OF THE HEART

GENERAL INSPECTION

The examination of the heart should be supplemented by a careful inspection of the position of the patient, the face, the eyes, the neck, the entire chest and the upper abdomen. "More mistakes are made by not looking than not knowing" (Jenner). The clothes should be removed from the upper part of the body and the patient placed in a good light. The observer should shift his position and that of the patient as may be necessary to get the light to strike at different angles and to secure the best illumination of certain portions of the chest, such as the area below the left breast where the cardiac impulse is usually visible, and the second right intercostal space where the pulsation of aortic aneurisms is most often seen. "Good eyes are the physician's best tools, but it is not merely acuteness of vision, though this is important, but it is the educated seeing eye which is only to be had by careful training" (Osler).

In severe cardiac insufficiency the patient is often unable to lie down (orthopnea). (Fig. 20.) Marked cyanosis is not as common in heart disease as it is generally thought to be, and is rarely seen in cases of the senile heart. Slight cyanosis is frequent in mitral stenosis. It sometimes gives the lips a color that is deeper red than normal, but hardly a blue shade. The subjects of early cardiosclerosis often look remarkably well, and their faces present the picture of vigorous health. There may be slight pallor in early arteriosclerosis due to a local anaemia. More advanced arteriosclerosis usually causes a peculiar whiteness of the face in marked contrast to the redness of the lips. This pallor is due to thickening of the small blood vessels and is the distinctive feature in the arterial facies.

The existence of an arcus senilis should be noted. This is a white opaque zone in the cornea near the junction with the sclera, and develops frequently in middle age. It is due to hyaline degeneration of the connective tissue fibres, associated with the deposit of lime salts in the cornea near the edge of the sclera. This condition is attributed to senile changes in the blood-vessels encircling the cornea. We should look, also, for in-

equality of the pupils which frequently occurs in aneurism of the thoratec aorta though generally the result of some cerebral affection and very often of luetic nature.

The expression should be observed and the character and rate of respiration. The breathing in cases of cardiac weakness often becomes deeper after the exertion of talking. Cheyne-Stokes or periodic breathing

Fig. 20.—Orthopnea in severe cardiac weakness. The half-sitting position is assumed. The patient is leaning back on his elbows. In the neck the contracted right sternocleidomastoid is visible. The hands and abdomen are swollen with fluid (edema). (Lüdke and Schlayer, "Lehrbuch der pathologischen Physiologie," J. A. Barth.)

is an indication of serious cardiac weakness and hence its presence should not be overlooked. It may disappear during conversation and thus escape detection. Its existence can often be discovered by having the patient relax, close the eyes and keep silent for a few minutes.

The temporal arteries are often tortuous and prominent even in the early stages of arteriosclerosis. In severe aortic insufficiency a slight jerking of the entire head may occur synchronously with the arterial pulse. More frequently in this disease there is a marked throbbing of the carotid arteries. When the throbbing is visible as high as the hollow between the sternomastoid muscle and the ramus of the jaw, it is of diagnostic significance. A pulsating deep jugular vein is sometimes mistaken for

the carotid but the difference can be recognized by the touch: the jugular pulsation is better seen than felt, while the carotid pulsation is better felt than seen.

Distention of the jugular veins when the patient is recumbent may occur normally, but if present when in the erect posture it is pathological. (1) It may occur as the result of cardiac weakness. If so, the veins will be partly emptied momentarily by a deep inspiration. In heart disease distention of the veins of the neck is a reliable sign of engorgement of the right auricle. (2) It may be due to pressure on the superior vena cava or both innominate veins by an aortic aneurism or mediastinal tumor. If the venous engorgement is due to pressure, inspiration will have no effect in reducing the distention. If only one jugular vein is distended it may be the result of pressure on one innominate vein. In distention of the jugulars due to heart disease there often occurs a marked venous pulse and suffusion of the face is common.

The study of the jugular pulsation yields important information regarding the right ventricle. Even in health some movement of the jugular bulb and the deep jugular vein above it can usually be seen, when the patient is lying down. Normally three waves can be made out. They can be roughly timed by placing a finger on the carotid artery and noting with the eye the relation of the jugular waves to the carotid pulsation. For careful study a graphic record is necessary.

The size of the thyroid should be determined. Hyperthyroidism, associated with enlargement of the gland, is frequently the cause of functional alterations in the heart—the thyroid heart. A downward movement of the thyroid cartilage of the larynx and a pulsation of the anterior wall of the trachea can be seen sometimes, but more often felt. It is produced most commonly by the pulsations of a deep-seated aortic aneurism and is called the tracheal tug. By grasping the cricoid cartilage with the thumb and forefinger and exerting gentle upward pressure on it the downward pull of the pulsating aorta on the larynx can often be felt when no movement is visible. (Oliver's sign of aneurism.)

1. **The Form of the Thorax.**—This may sometimes throw light on the nature of the disturbance of the circulation. In emphysema of the lungs the chest assumes permanently the position or form of inspiration though in an exaggerated degree. This is the most characteristic feature of the emphysematous chest. (Fig. 21.) In inspiration the lower part of the normal thorax becomes more full, so in emphysema the chest does not taper from above downward as does the normal chest but becomes barrel shaped. The sternal fossa is deep and the clavicles and sternomastoid muscles stand out prominently. The back is rounded. In this

disease there is often weakness of the right ventricle due to increased resistance to the circulation of the blood through the lungs. Kyphoscoliosis produces a marked deformity of the chest. (Fig. 22.) The spine is curved in its anteroposterior diameter with the convexity of the curve behind (kyphosis) and it is also curved laterally (scoliosis). The ribs bulge on one side of the front of the chest and are depressed on the

FIG. 21.—Emphysematous thorax, paralytical thorax, normal thorax. (Brugsch und Schittenhelm, "Lehrbuch klinischen Untersuchungsmethoden," Urban and Schwarzenberg.)

other side. Posteriorly on the side of the concavity of the scoliosis the back is flattened; on the other side the back bulges. Kyphoscoliosis, like emphysema, often results in circulatory symptoms due to failure of the right ventricle. Both of these diseases interfere with the normal impulse of the heart against the chest wall, and alter the percussion outline even when the heart is of normal size. More easily overlooked than these deformities is the chest in which the anterior and lateral portions of the ribs form a sharp angle in the mammary or axillary region so that the cross section of the chest becomes more or less quadrilateral. A chest of this form influences the findings on palpation and percussion of the cardiac impulse and its existence must be recognized. It occurs particularly in children, but occasionally is seen in adults.

2. **Bulging of the Cardiac Region.** (*Voussure.*)—Great enlargement of the heart occurring in early life, while the bones are soft, produces a marked prominence of the chest well to the left of the lower half of the sternum and sometimes of the sternum itself. A large precordial

effusion in childhood may have the same effect. This bulging never develops in adults, even when cardiac hypertrophy occurs, because the thoracic wall is hard and does not yield to the pressure. Its existence after

FIG. 22.—Kyphoscoliotic thorax. (Morawitz "Klinische Diagnostik innerer Krankheiten," F. C. W. Vogel.)

childhood is evidence that the enlargement of the heart is of long duration, and one should consider the possibility of congenital heart disease.

3. **Pulsation in the Suprasternal Notch.**—This may arise in the arch of the aorta, but more commonly has its origin in the innominate or right common carotid. It may occur (1) in aortic insufficiency and sometimes in this condition the pulsating vessel can be seen and felt; (2)

aneurism of the transverse arch or innominate artery; (3) simple dilatation of the aorta in arteriosclerosis; (4) neurotic conditions may produce a dynamic dilatation of the aorta and other arteries.

4. Bulging and Pulsation in the Second Right Intercostal Space Near the Sternum.—This is the most common situation of the pulsation produced by aortic aneurisms. Sometimes in aneurism a large swelling projects from the anterior chest wall to the right of the sternum. Again an aneurism may produce only a heaving impulse of the upper part of the sternum that is easily overlooked unless the light falls at the right angle and the inspection thorough. When localized and of any extent, the pulsation is visibly expansile. It may also occur in aortic insufficiency and rarely in association with mediastinal tumors.

5. Pulsation in the Second Left Intercostal Space Near the Sternum.—(1) Aneurisms of the aorta and mediastinal tumors often produce a pulsation in this region. (2) It may be transmitted from the normal pulmonary artery, when the overlying lung is pushed or drawn away and the vessel left uncovered and in contact with the anterior chest wall. This may occur in mitral stenosis when a greatly dilated left auricle pushes back the lung or more rarely in chronic phthisis from contraction of the lung. (3) The dilated left auricle in mitral stenosis may itself give rise to the pulsation. This has been shown by graphic records (Rauntenberg). (4) Dilatation of the pulmonary artery is rare (congenital heart disease) but may be the cause of pulsation in this region.

6. Diffuse Pulsation of One Side of the Chest.—This may result from a deep-seated aneurism or be due to pulsations of the heart transmitted through a collection of pus in the pleura (empyema) or serous fluid (pleurisy with effusion), or air in the pleural cavity (pneumothorax), or through a solidified portion of the lung (pneumonia). It may also occur in tumor of the lung or mediastinum or in chronic mediastinitis, as these conditions may lead to the transmission of the cardiac impulse to a large portion of the chest wall.

INSPECTION AND PALPATION OF THE CARDIAC REGION

1. The Normal Cardiac Impulse.—In the fifth left interspace between the parasternal line and the mammillary line there can often be seen in healthy subjects a slight outward thrust of the chest wall with each beat of the heart. The location of this cardiac impulse gives important information regarding the size and position of the heart in health and in

disease. The heartbeat can be best seen in women after the breast has been drawn upward and to the right.

To locate the cardiac impulse the hand should be placed on the surface of the chest below the nipple. The area of impulse thus determined should be carefully palpated with the tips of the second and third fingers. The impulse is usually more readily felt than seen. Normally it can be covered with the tips of one or two fingers. The outermost and lowermost point of definite impulse should be carefully located, as it is an index of the size of the heart. This has been generally called the apex-beat.

The cardiac impulse is usually better seen and felt with the patient in the recumbent than in the erect position. By turning on the left side and exhaling the impulse usually becomes more prominent and it may be made out by this procedure only.

In children up to the tenth year the impulse is regularly in the fourth interspace in or just outside the nipple line. This is due to the high diaphragm which causes the heart to assume a more horizontal position, shifting the impulse upward and outward. In women it is often in the fourth interspace. In old age it may be in the sixth interspace. The position of the impulse is often located with reference to the mammillary line but this is too variable in women to be used as a landmark. It is far better to measure the distance of the impulse from the median line. In health it varies with the size of the subject, but the outer portion, that is the so-called apex-beat is usually in adults 8 to 10 cm. from the median line.

Up to the twentieth year the cardiac impulse can be felt in practically every chest in health. With increasing age it can be less readily palpated, and by the age of 50 it can be felt in only about 40 per cent of people. In old age it becomes again more frequently palpable. (Guleke.)

This variation in the palpable impulse depends chiefly upon the intimate relation (1) between the position of the diaphragm and that of the heart and (2) the force of the systolic impact of the heart against the chest wall. The cardiac apex is wedged in between the dome of the diaphragm and the anterior chest wall. The greater the arching of the diaphragm, the greater will be the resistance it offers to the increase of the anteroposterior diameter of the heart during systole, and the geater will be the outward thrust of the heart against the chest wall.

With increasing age there is a loss of elasticity in the lungs with an increase in volume. As a result the diaphragm becomes flatter. This increases the angle between the diaphragm and the chest wall and the resistance offered by the diaphragm to the systolic increase of the anteroposterior diameter of the heart is lessened. As a result the palpable

cardiac impulse will become weaker or disappear. At the same time this change in the position of the diaphragm occurs the layer of lung between heart and chest wall increases, as the lung becomes more voluminous, and the chest wall becomes more rigid and thicker.

In old age the wasting of the soft parts causes the chest wall to become thinner. This, together with senile hypertrophy and dilatation of the heart, explains the increased frequency of a palpable cardiac impulse in the aged.

The location of the impulse is affected by the shape of the chest. If the chest is broad and short and the diaphragm high the impulse may normally be in the fourth space and further out than usual. While in a long narrow chest with a low diaphragm, it may be in the sixth space, and nearer the median line.

The position of the cardiac impulse shifts with the position of the subject. Lying on the left side causes the impulse to move 1 to 3 cm. to the left. It may then be, in some individuals, nearly as far out as the midaxillary line. To this unusual displacement of the heart the term cor mobile is applied. There is less movement to the right produced by lying on that side, but sometimes this position causes a disappearance of the impulse. An ordinary inspiration does not affect the cardiac impulse but a deep inspiration may cause it to descend an interspace. When the area of impulse is covered by a rib an inspiration may bring it within reach of the eye and hand.

2. The Cause of the Cardiac Impulse.—The anterior and lower portion of the heart lies in the angle formed by the anterior wall of the chest and the diaphragm. In diastole the heart muscle is flabby and the lower end rounded. Its cross-section is elliptical with the long diameter from right to left and the short diameter from before backwards. With the beginning of systole the muscle hardens, the lower end rotates to the right. The shape of the cross section changes from an ellipse to a circle. This increases the antero posterior diameter at the same time that it decreases the lateral diameter. A bulging occurs on the anterior surface of the left ventricle above the apex and just inside the left border, and this strikes against the chest wall producing the outward thrust. The impulse occurs during the early part of systole—the isometric phase—when both the atrioventricular and the semilunar valves are closed. It is not influenced apparently by the amount of blood in the heart. In fact, animal experiments show that an empty heart can produce a strong beat. The impulse is not produced by the apex of the heart and hence the term apex-beat, although generally used, is not correct. Orthodiagraphic studies show that the outward thrust of the chest wall is usually

an interspace above the true apex and inside the left border of the heart.

3. **Displacement of the Cardiac Impulse.**—Even when the heart lies in its normal position and its size is unaltered, deformity of the chest may cause a displacement of the impulse from the usual situation, as is seen in kyphoscoliosis which changes the curves of the ribs and the position of the sternum. But usually a displacement of the impulse is due to a change in the position or size of the heart.

Displacement Upward.—This occurs in any condition which forces the diaphragm upward, owing to the close relation between the heart and the diaphragm. In ascites, meteorism, abdominal tumors, and in pregnancy the impulse may be pushed up one or two interspaces.

Displacement Downward.—When the diaphragm occupies a low position the heartbeat will be lower than the fifth interspace. In emphysema, due to increased volume of the lungs and the resulting change in the shape of the chest and the low position of the diaphragm, the cardiac impulse may be so depressed that it occupies the epigastric region. Dilatation of the left ventricle shifts the impulse downward and outward.

Displacement to the Left.—Right sided pneumothorax or right sided pleuritic effusion pushes the heart to the left, and causes greater displacement of the heart than when these space-reducing processes occupy the left side of the chest. (Morawitz.) The heart impulse is shoved a little upward because depression of the right half of the diaphragm raises the left half of the diaphragm and with it the heart. Fibrosis of the left lung or shrinkage of the left lung from any cause results in a movement of the impulse to the left. The most common cause, due to the heart itself, is enlargement of the left or right ventricle or both.

Displacement to the Right.—This is produced by left sided pneumothorax or pleuritic effusion and by disease in the right lung leading to its reduction in size. The cardiac impulse may under these conditions be entirely to the right of the sternum. Chronic lung disease can so displace the heart that the impulse is felt in the right nipple line.

The heart is not pulled over by pleuritic adhesions either to the right or left side as generally taught, but is pushed over by the healthy lung. When one lung becomes smaller from any cause the other lung becomes compensatorily larger, pushes the mediastinum and the heart toward the shrunken lung, and thus fills the space that the diseased lung occupied before its size was lessened.

4. **Increase of the Area of Cardiac Impulse.**—A high diaphragm, a tumor of the lungs or posterior mediastinum, and especially aneurism of the descending aorta, by pressure on the heart from behind increases the extent of the contact of the heart with the anterior chest wall. Retraction

of the left lung in chronic phthisis or as a result of pleuritis leaves a greater portion of the heart uncovered and this increases the area of visible and palpable cardiac impulse. It may extend as high as the second interspace and laterally from the sternal border to the left mammillary line. Enlargement of the left ventricle leads to a more extensive impulse, especially downward and outward. Enlargement of the right ventricle increases the area upwards and outwards and also toward the median line. When the dilatation and hypertrophy of both sides of the heart are extreme the visible cardiac impulse may extend over the entire front of the chest from the second costal cartilage on each side of the sternum downward, and laterally from one axillary line to the other (Gerhardt).

5. **Increase in the Force of the Cardiac Impulse.**—Normally, as already stated, the impulse is a slight tap and even this may be absent. As a result of exercise or excitement the force of the impulse may increase considerably. It is a quick sharp stroke and is usually associated with an increase of the heart rate. In cases of cardiac neurosis or the effort syndrome the increased force of the cardiac impulse may be the chief physical sign on examination of the heart. A similar increase in the force of the impulse occurs in fever and in hyperthyroidism. In hypertrophy and dilatation of the heart the force of the impulse may be greatly increased. In fact this is the most characteristic sign of cardiac hypertrophy. Hence the strength of the impulse should be measured as carefully as possible, and to do so requires training. It is important to estimate the force of the impulse in every heart examined and it is well to record it on the case report. The character and force of the impulse may be more accurately gauged by the ear and the side of the face resting on the chest. This was recognized by Bayle and it was while Laennec was attempting to estimate the force of the cardiac impulse that he discovered auscultation. The impulse may be (1) increased in force, (2) much increased or throbbing and (3) heaving. The separation into three grades, of hearts exhibiting an increase in the force of the impulse, was made years ago by Bamberger and it has proved useful in practice. He characterized the throbbing impulse (*Erschütterung*) as follows: The hand placed flat on the cardiac region feels a powerful thrust like a blow made by solid body. The rapidity of the cardiac contraction is regarded as the cause of the throbbing impulse. As soon as the movements of the heart that cause the impulse are slowed the throbbing ceases (G. Weber). In fever, in excitement, after physical exertion, in cardiac neurosis and in hyperthyroidism the force of the impulse may be increased or much increased, but it is rarely if ever heaving. Sometimes in cardiac neurosis the impulse may be very forcible at one examination, though

never continuously so, and only when the heart rate is increased. In hypertrophy of the heart any one of these stages may be present, but a heaving impulse is common. The character of the impulse often differs from that of a nervous heart. The rise of the chest wall under the palpating finger is more gradual and the outward thrust more sustained in hypertrophy. It gives the impression through the finger that a curve of the impulse in hypertrophy would be plateau-shaped while the impulse due to excitement would have the form of a peak with a sharper ascent and descent and with no plateau.

The term heaving is reserved for an impulse so strong that it cannot be obliterated by the palpating finger even when much force is applied. Often the ribs as well as the interspaces are thrust forward. The movement may be slow or rapid. The force of the impulse is no index to the strength of the heart. It may be heaving when heart failure is marked. The contrast between a very forcible cardiac impulse and a weak radial pulse in cardiac insufficiency may be striking.

6. Decrease in the Force of the Cardiac Impulse.—The impulse may be feeble or absent even when the heart is strong. In obesity and in emphysema the cardiac impulse is usually absent, and in healthy adults with increasing age the impulse becomes less marked owing to the low position of the diaphragm and the loss of elasticity in the lungs. The disappearance or weakening of the impulse during the course of a fever often accompanies increasing cardiac weakness. Its disappearance under observation is also of value in the recognition of pericardial effusion. In pericardial effusion the only cardiac impulse may be over the body of the heart in the third or fourth left interspace near the sternum. The contrast in size between the small area of visible and palpable cardiac impulse and the wide area of dullness helps to distinguish pericardial effusion from great dilatation of the heart as does the wider zone of dullness in pericardial effusion between the left border of cardiac impulse and the left border of the heart.

7. Changes in the Location of the Cardiac Impulse in Dilatation of the Heart.—Dilatation of the heart shifts the position of the impulse downward and to the left and increases the area of visible and palpable impulse. For some reason as yet unknown the hypertrophy and dilatation associated with valvular disease usually produces a wider and more forcible impulse than hypertrophy and dilatation due to cardiosclerosis. In great enlargement of the left ventricle the lowermost and outermost portion of the impulse is lower than in enlargement of the right ventricle. The latter causes the apex impulse to move out toward the axilla more than it moves down toward the costal border. In aortic insufficiency

there is often great hypertrophy and dilatation of the left ventricle and the cardiac impulse frequently occupies the seventh or eighth interspaces. In mitral stenosis there may be marked enlargement of the right ventricle with no impulse present below the fifth interspace. An impulse extending outward from the left border of the sternum is often seen in dilatation of the right ventricle. A pulsation in the third or fourth interspace near the sternum may arise in the conus arteriosus of the right ventricle. In great dilatation of the right ventricle an impulse is seen to the right of the sternum, usually in the fourth interspace, but sometimes higher.

8. Systolic Retraction Replacing the Systolic Outward Thrust.—This occurs in chronic mediastinopericarditis, a condition in which firm adhesions bind the heart to the pericardium and this to the ribs and sternum. There may be a forceful retraction of the ribs over the heart and sometimes of the lower end of the sternum. Even in the region of the apex-beat a withdrawal of the soft tissues during systole may occur in place of the normal protrusion. The systolic depression of the sternum rarely if ever occurs except in adherent pericardium. In some instances of cardiac enlargement where the thrust is strong and high there may be seen inside the area where the chest wall is driven out by the systole a slight indrawing of the chest wall. This systolic retraction is without significance and is due to the increase of the negative pressure produced by the powerful contraction of the enlarged heart.

The cardiac impulse produced by the right ventricle, which is marked in cases of mitral stenosis, is usually an outward thrust like that caused by the left ventricle. Under certain conditions there may be a systolic retraction over the right ventricle giving rise to the "inverted cardiogram"

FIG. 23.—Simultaneous tracings of the apex-beat and of the radial pulse. The rise in the apex tracing resembles the usual characters present in a tracing of the apex-beat due to the left ventricle. On analysis it is found that the elevation is during the diastole, and the fall (E) during the systole, of the ventricle. (Mackenzie, "The Study of the Pulse," Young J. Pentland.)

(see Fig. 23). This change in the character of the impulse caused by the right ventricle is probably due to free regurgitation of blood through the tricuspid valve into the superior and inferior vena cava—tricuspid insufficiency (Lang).

9. **Diastolic Outward Thrust or Back-Stroke.** (Hope)—In addition to the systolic protrusion a second outward movement sometimes takes place in early diastole. This may occur in aortic insufficiency after the heart muscle has weakened and is often accompanied by a protodiastolic gallop rhythm. It is also distinctly present in some cases of chronic mediastinopericarditis.

10. **Palpable Shock Associated with the Second Pulmonic Sound.**—This is felt in the second left interspace near the sternum when the second pulmonic sound is very loud and is due to unusually high pressure in the pulmonary artery. The vibrations that cause the sound are so strong that they are transmitted through the chest wall to the palpating hand. This would not occur if the orifice of the pulmonary artery was not in such close contact with the solid tissues of the anterior part of the chest. No shock is felt over the aortic area even when the aortic second sound is loud and ringing because between the anterior surface of the chest and the aortic ring there are three media to traverse,—air in the lungs, fluid in the blood-vessels overlying the aorta, and the solid tissues of the chest wall,—and these so damp the vibrations that they cannot be palpated.

11. **Friction-Rubs and Thrills.**—In cases of acute fibrinous pericarditis a rub can often be felt over the heart due to roughness of the pericardium as a result of fibrinous deposits. Over different portions of the cardiac area fine vibrations are transmitted to the hands, in certain types of valvular disease of the heart, associated either with the systole or the diastole. Near the so-called mitral area or apex region—that is, the fifth left interspace inside the nipple line—there is frequently felt in mitral stenosis a diastolic thrill that has been aptly compared to the sensation given to the hand when placed on the back of a purring cat. This thrill is so distinctive of mitral stenosis that when typical the diagnosis of this lesion is justifiable. It is as Dr. Osler used to say the only valvular disease that can be diagnosed on palpation. Caution is needed, however, as in many cases of cardiac neurosis with an increased cardiac impulse and in healthy individuals under excitement a pseudothrill is present at the mitral area. The vibrations communicated to the hand by the excited heart are coarser and less numerous than those of a true thrill and should not confuse anyone who has ever felt a well-marked thrill in mitral stenosis. The pseudothrill is furthermore almost always systolic in time and is associated with a rapid, forcible heart action. It is often nothing more than a quivering impulse. Over the mitral area a systolic thrill is sometimes felt in mitral insufficiency, but is rare, and less intense than the thrill of mitral stenosis. In the second right inter-

space near the sternum—aortic area—a systolic thrill is usually found in aortic stenosis. It is a characteristic physical sign of this lesion and, unless present, the diagnosis is rarely justifiable. A diastolic thrill over the aortic area occurs in aortic insufficiency, but it is not common. A systolic thrill over the pulmonic area—second left interspace near the sternum—occurs in pulmonary stenosis; a rare condition and one of congenital origin. Another rare lesion is tricuspid stenosis. When present it may produce a diastolic or presystolic thrill which is felt near the lower end of the sternum over the fourth left costal cartilage.

EPIGASTRIC PULSATION

This is frequently due to the pulsations of the abdominal aorta being transmitted to the abdominal wall, especially in thin persons and in those with lordosis of the spine. In nervous individuals it may be remarkably strong and has often been mistaken for an aneurism. On deep palpation the aorta can be felt against the spine. If the sternum is short or the diaphragm low an epigastric pulsation of cardiac origin may occur even when the heart is normal. In emphysema the cardiac impulse may be present in the epigastrium and not elsewhere.

When the right ventricle is dilated and hypertrophied and the diaphram flattened an epigastric pulsation is common. It is usually seen and felt in the left xiphocostal angle and below the tip of the xiphoid cartilage. Occasionally the impulse occupies the entire area between the two costal borders. The impulse is generally an outward thrust similar to that over the cardiac area. It may, however, be a retraction during systole instead of a protrusion. This possibly occurs in association with tricuspid insufficiency.

The presence and force of an epigastric pulsation bear no relation to the degree of hypertrophy and dilatation of the right ventricle (Lewis).

PERCUSSION OF THE HEART

Introduction

Percussion of the heart is of great value in determining the size of the organ but it is not a method of precision. There are errors and difficulties in marking out the exact size that are unavoidable, but there are other errors common in practice that can be eliminated by learning the proper technic for outlining the heart by percussion. The results obtained by percussion need to be considered in relation to the shape of

the chest, the position of the diaphram and the condition and position of the lungs before they can be properly interpreted. Students should realize at the outset of their study of clinical medicine that cardiac percussion is the most difficult of the diagnostic procedures they will be called upon to employ in physical diagnosis. By use of right methods of percussion, practised whenever the opportunity presents, and with results controlled by the orthodiagraph, any student should be able to acquire sufficient skill to make the percussion of the cardiac outline a real aid in the diagnosis of cardiac disease.

The difficulty of marking out the cardiac outline is shown by the fact that until recently the writers of standard textbooks differed widely as to the position of the right and left borders of the normal heart as determined by percussion. This led to much confusion and uncertainty. The introduction of the orthodiagraph, which gives the exact size of the heart in the frontal plane, made possible a critical study of cardiac percussion. The methods of percussion now used are those that have been shown by comparison of percussion findings with orthodiagrams to give the most trustworthy results.

Inherent Difficulties in Cardiac Percussion

Only a portion of the anterior surface of the heart is in direct contact with the wall of the chest; the remainder is covered by the lungs. The right border of the heart which is formed by the right auricle is at a considerable distance, 3 to 8 cm., from the surface of the chest. (Fig. 24.) A thinner layer of lung-tissue separates the left border of the heart from the chest wall. If the chest is narrow with sharp, sloping sides, the left border of the heart as determined by percussion will be outside the nipple line and often in the axillary region even when the heart is of normal size. Even if the chest is normal a strong percussion-stroke used in determining the left border of the heart will give a dull note far outside the vertical projection of the left border on the anterior surface. The normal right border of the heart usually lies 4 to 5 cm. to the right of the median line and 1 to 2 cm., or a finger's breadth, beyond the right sternal border. That it is not easy to locate this border by ordinary percussion is evident from the statement in some textbooks of physical diagnosis that the right border of cardiac dullness is normally at the left sternal border.

The vibrations produced by a percussion-blow on the chest spread in all directions. According to Weil the depth of the lung-tissue set in vibration does not exceed 5 or 6 cm., while Martini maintains that if a solid structure such as the heart is covered by a layer of lung-tissue over

4 cm. thick, its presence will not be revealed by percussion. A thin layer of air-containing lung not over 3 cm. in thickness situated in front of the heart or other solid structure yields a higher, shorter, and consequently a duller note on percussion than a thicker layer of lung. This physical

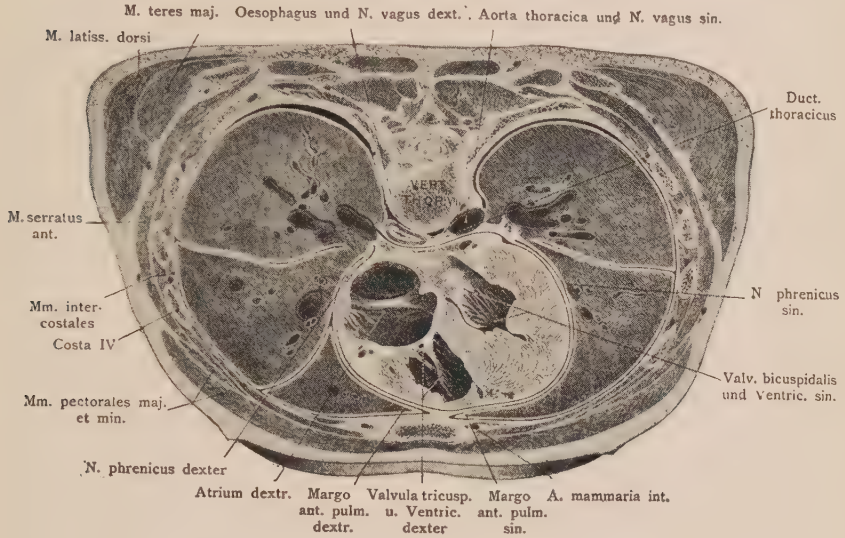


FIG. 24.—Horizontal section through the chest at the level of the eighth thoracic vertebra. Note the distance of the right border of the heart from the anterior surface of the chest. (Corning, "Lehrbuch der topographischen Anatomie," J. F. Bergmann.)

property makes it possible to percuss out the "relative" cardiac dullness. It is evident, however, if these statements are true, that when the right border of the heart is more than 4 cm. below the surface of the chest it cannot be located by percussion. Relative cardiac dullness in comparison to the resonant sound over the lung-tissue is due to the replacement of a part of the air-containing tissue in the percussion zone, by the airless tissue of the heart. But it takes a considerable reduction in the volume of air-containing pulmonary tissue in the percussion zone in order to dull the percussion-note.

The boundary of relative cardiac dullness will vary somewhat with the force of the percussion-stroke. The term strong percussion is relative. One examiner using strong percussion will employ a lighter stroke than a second examiner. The result is shown in Fig. 25. Examiner A using the lighter blow sets in vibration a smaller vibration zone which does not include any of the airless heart. This is indicated by a solid line. The sound produced is as resonant as over the lung elsewhere. Exami-

ner B using a heavier stroke sets in vibration a larger area, marked by the interrupted line, and the resonance is less owing to the presence of heart-tissue within the area than it would be if composed entirely of air-containing lung-tissue. Examiner B will mark the point percussed

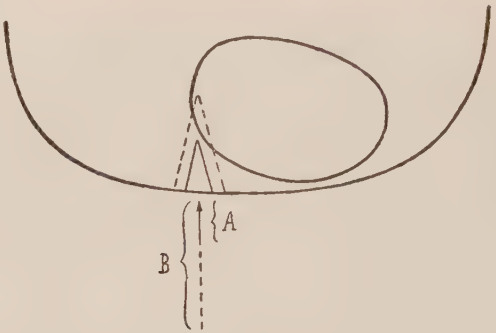


FIG. 25.—This diagram illustrates the variation in the position of the right border of relative cardiac dullness dependent on the force of the percussion-stroke. Two examiners are employing "strong" percussion, but the force of their blows vary. Examiner B uses the heavier stroke represented by the full length of the arrow. The heavier stroke produces the larger percussion zone which includes in addition to lung-tissue a portion of the heart. The note is dulled and the examiner takes this point on the skin as the right border of the heart. Examiner A using a lighter stroke, represented by the short uninterrupted line of the arrow, sets a smaller area of tissue in vibration,—shown diagrammatically by the smaller v shaped area. This does not include any portion of the heart in addition to the lung-tissue. The note is resonant. Examiner A concludes that the point percussed is beyond the right border of the heart With his lighter percussion stroke the right border of dullness will lie some distance nearer the middle line. (Schmidt and Luetje, "Klinische Diagnostik innerer Krankheiten," F. C. W. Vogel.)

as the border of relative cardiac dullness while A will get dullness with his lighter stroke at some distance within this point. Furthermore, the size of the vibration area depends in part on the pressure with which the finger used as a pleximeter is applied to the skin of the chest. The firmer the pressure the larger the area of tissue set in vibration. As the sound elicted varies with the force of the blow and the pressure exerted by the finger, it is evident that the percussion of relative dullness involves a large subjective element as the intensity of the blow and the pressure of the finger cannot be measured. Hence these variable factors make impossible absolute certainty that the relative heart dullness corresponds to the size of the heart in the frontal plane. The same examiner must use percussion-strokes of varying strength in outlying different borders of relative cardiac dullness on the same subject, according to the thickness of the chest wall. A heavier stroke is necessary for the right border than for

the lower part of the left border and a heavier one for the upper border and upper part of the left border of the heart. The fat and muscle layers are thicker in the region of the breast especially in women. In examining subjects with a thick layer of subcutaneous fat over the thorax the strength of percussion must always be stronger than in the case of thin individuals. Increase of muscular tension also affects the results of percussion by impairing the resonance of underlying tissues. The chest of a crying child cannot be accurately percussed for this reason and not solely because the cry of the child obscures the percussion sound.

The sternum acts as a pleximeter. The impairment of resonance produced by the inclusion of its right border in the vibration area is often wrongly interpreted as dullness due to the right border of the heart.

It might seem from this consideration of the difficulties of percussing the true cardiac outline that a strong percussion-stroke would be preferable to a light one. This, however, is not so. The difference between the sound produced over the air-containing lung and the airless tissues of the heart is more sharply differentiated by light percussion. A percussion-stroke of even moderate force over the lung brings out the fundamental note of the hemithorax. Hence in determining the limits of relative cardiac dullness it is essential to use a stroke so light that it will not call forth the resonator property of the thorax.

Absolute Cardiac Dullness

This is also known as the superficial or small cardiac dullness. Its area corresponds approximately to that part of the anterior surface of the heart that is not covered by the border of the left lung. On light percussion this area yields a very dull sound; in fact on very light percussion passing inward from the lung to the heart the resonance yielded by the lung disappears and no sound is heard. This transition from some sound to no sound is one that can be brought out by the beginner who may be quite unable to distinguish differences in the pitch or duration of sounds elicited by percussion. The area of absolute dullness can be determined with ease, and the correct method quickly learned. To mark out the relative cardiac dullness correctly is difficult. The proper technic requires skill and experience. This difference is due to the fact that the ability to outline the absolute dullness depends far less on the examiner's interpretation of what he hears than does the percussion of the relative or deep cardiac dullness. The student should begin the practical study of cardiac percussion by acquiring the ability to mark out the area of absolute cardiac dullness. Many practitioners who now make no use of

cardiac percussion, because they have no confidence in their ability to determine even roughly the area of relative cardiac dullness, will find that they can easily learn to percuss out the area of absolute cardiac dullness and that it will be a great aid to them in the routine examination of the heart. This is due to the fact that the size of this area is greatly influenced by the size of the heart. If the lungs and pleura are normal, the heart, as it enlarges, and also the pericardial sac, as it becomes distended with fluid, push back the border of the lung, both to the right and to the left, and this results in an increase in the area of absolute cardiac dullness. The size of this area is usually a good index of the size of the heart or of the pericardial sac. (Fig. 26.)

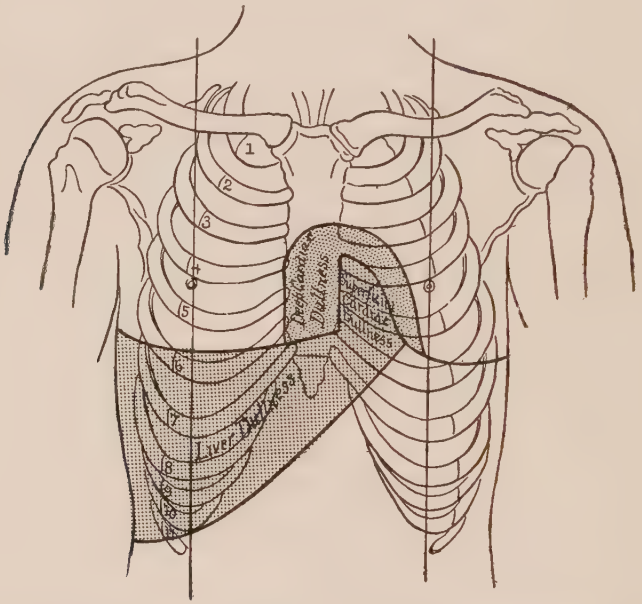


Fig. 26.—Absolute (superficial) and relative (deep) cardiac dullness under normal conditions. (Sahli, "Diagnostic Methods," W. B. Saunders Company.)

The area of absolute dullness is not exactly that portion of the heart in contact with the chest wall. It does not extend normally over the sternum although the heart is uncovered by the lung to about the median line. This is due to the property of the sternum to act like a large pleximeter, the upper portion of which is in contact with air-containing lung. As a result even a light percussion-stroke on its lower part yields a resonant note. The area of absolute cardiac dullness extends to the left beyond the margin of the left lung, because the lingula is too thin to affect the percussion sound.

The area of absolute dullness gives more direct evidence regarding the position of the lung borders than it does of the size of the heart. If the borders of the left lung are retracted, as a result of fibrosis or other disease, the area of absolute cardiac dullness will increase although the size of the heart is unchanged. If the lungs become more voluminous, as in emphysema, more of the anterior surface of the heart will be covered by the air-containing pulmonary tissue and the area of absolute cardiac dullness will be diminished or even obliterated. Usually, when the heart enlarges, the borders of the lung are pushed back and the area of absolute dullness increases in extent. Pleural adhesions may so fix the borders of the lung in the neighborhood of the heart that they do not move even when the heart increases greatly in size. Then the area of absolute cardiac dullness will remain unchanged and thus give no evidence of the cardiac enlargement.

Much fat over the chest makes the percussion of the area of absolute cardiac dullness untrustworthy. Large breasts also make it unsatisfactory. The left breast should be drawn upward and supported by the hand of the subject during percussion. Gas in the stomach may replace the absolute cardiac dullness by a tympanitic sound.

It thus becomes evident that the examiner should not be content to determine the area of absolute cardiac dullness alone, but should attempt to mark out, in addition, the area of relative cardiac dullness. Before percussing the absolute cardiac dullness, carefully determine the lower border of pulmonary resonance in the right mammillary line using light percussion. This reveals the position of the upper margin of the liver in contact with the anterior wall of the chest, and also that of the diaphragm. This lower boundary of the lung marks also the level of the lower border of cardiac dullness because the heart rests on the diaphragm.

In percussing the heart the examiner should observe the rule to pass from the region that yields a resonant sound to the one that yields a flat sound. In this case from the lung toward the heart and not vice versa. In the attempt to verify one's findings it is allowable to percuss from the heart to the lung. This procedure has recently been recommended by Edens.

Determine the right border of the heart by percussing from the right mammillary line toward the sternum in the first interspace above the lower right pulmonary border. The position of the examiner should be at the right of the subject. The plexímeter-finger is placed in the interspace and held parallel to the long diameter of the interspace. The finger should be lightly applied to the skin of the chest and a light percussion-stroke used. The head of the examiner should be in front of the chest

with the ear near the surface that is being percussed. Move the plexim-eter-finger to the left until the resonance is replaced by a sound that is absolutely dull, or until no sound is heard except the slight tap of the per-cussion-stroke on the finger. In similar fashion percuss the left border from without inwards beginning at a point beyond the left nipple line. The upper border is made out by percussing from the second interspace downwards just to the left of the sternal border. The limit of absolute dullness is determined at various points between the upper margin and the lower left border. The lower border can often be determined in the region of the apex, by percussing from below upward in the line of the left border of absolute dullness and at several points inside this line. The change on light percussion from the note of gastric tympany to the dull sound over the heart is often sharply marked.

Normally the right border of absolute cardiac dullness is at the left sternal margin and the left border at or just inside the apex-beat. The upper border is at the fourth left costal cartilage near the sternum. This point is connected with the left border in the region of the apex-beat by a line curved in its upper part, but running in a more vertical direction as it approaches the apex region. The transverse width of absolute dullness measured at the base of the area, that is the under side, is usually 6 to 8 cm. In children up to the tenth year the upper border may be as high as the third interspace or even the third left costal cartilage. In old people the upper border of absolute dullness is usually at the fifth costal cartilage.

Children have a relatively large heart in comparison with the size of the chest, and the area of absolute dullness is relatively somewhat larger. In old age and in emphysema the area is diminished. A high diaphragm brings more of the heart in contact with the chest wall and the heart in this position pushes the borders of the lung further to the side. The area of absolute dullness is thereby increased. If the diaphragm is low the heart falls backward and downward so that less of the surface is in contact with the chest wall and the area of absolute dullness will be lessened. Before concluding that an increased area of absolute dullness indicates cardiac enlargement the existence of pulmonary disease must be excluded and the position of the diaphragm determined.

If the borders of the lungs are fixed by adhesions or if the diaphragm is low, the greatest enlargement of the heart can exist without any increase in the area of absolute cardiac dullness (Gerhardt).

Enlargement of the left ventricle pushes the border of the lung to the left and upwards. This increases the absolute cardiac dullness to the left and from above downwards. Enlargement of the right ventricle dis-places the border of the right lung lying behind the lower sternum to the

right. This results in moving the right border of absolute dullness from the left sternal margin to the middle of the sternum or even to the right margin. The right boundary of absolute dullness instead of running vertically, as it does normally, follows an oblique line from the left sternal border downward and to the right toward the right sternal margin. Often the boundary has a step-like outline (Krönig's steps). (Fig. 27.)

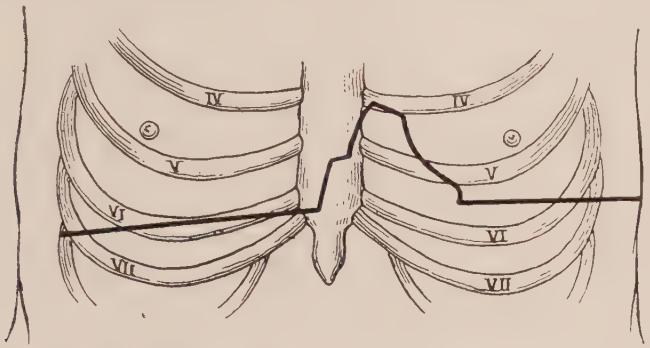


FIG. 27.—Step-like right border of the area of absolute cardiac dullness in dilatation of the right ventricle. (Krönig, Berl. klin. Wchenschr.)

The difficulty of percussing over the sternum due to its pleximeter-like property should again be emphasized. Pressure over the bone with one or two fingers outside of the area percussed will so damp its vibration that more trustworthy results will be obtained. The percussion-stroke over the sternum must always be light!

The recognition of the step-like border of absolute dullness behind the sternum described by Krönig is of diagnostic importance, because it indicates dilatation of the right ventricle which might otherwise escape detection. Definite increase of the area of absolute dullness to the right due to an enlargement of the right ventricle often occurs before there is any increase in the area of relative cardiac dullness to the right or before there is any bulging of the right border in the orthodiagram. Dilatation of the right ventricle often occurs without dilatation of the right auricle, which forms the right border of the heart, or without displacing the right auricle to the right. If the absolute dullness extends well to the right of the right sternal border it is usually the result of dilatation of the right auricle or due to pericardial effusion. An increase of absolute dullness to the right similar to that occurring in dilatation of the right ventricle is found in two other conditions. (1) In obese persons: an increase of fat in the epicardium and in front of the pericardium may produce absolute dullness over the lower sternum when the heart is of normal size.

(2) In men with very short and broad chests: the heart lies nearly horizontal and there is a small area of absolute dullness to the right of the median line at the lower end of the sternum (Matterstock).

After marking out the area of absolute cardiac dullness it is well to determine the movability of the pulmonary borders in the cardiac region. Have the person take a full inspiration and then hold the breath. If the lung borders are not adherent the area of absolute dullness will either greatly diminish in size or disappear and then a resonant sound will be obtained on light percussion over the entire cardiac area.

Relative Cardiac Dullness

This is also known as the deep and sometimes as the large cardiac dullness. It corresponds to the true size of the heart in the transverse diameter when accurately determined. Relative cardiac dullness is of greater value than the absolute dullness, in cardiac diagnosis, because the latter gives simply the area uncovered by the lungs; but it is far more difficult to acquire the ability to make out the relative dullness than the absolute dullness. Whenever possible, the student should check his cardiac percussion outlines with the exact boundaries determined with the orthodiagraph. Some recent writers, notably Lewis and Wilson, hold that the value of percussion in determining the size of the heart is overestimated. Others, especially Moritz, Dietlen and Goldscheider, maintain that by the use of special methods the true cardiac outline can be marked out by percussion in the majority of cases almost as accurately as with the orthodiagraph. I believe that the truth lies midway between these opposing views. I am sure that if physicians and students trained themselves more thoroughly in cardiac percussion, using a proper technic, and if, in all cases, the absolute as well as the relative dullness was determined, they would find that for them the value of cardiac percussion, with all its limitations, was greatly increased. Slight changes in the size of the heart certainly cannot be determined; and I agree with Wilson that, "valuable as it is as a clinical method, it is almost valueless where accuracy is essential." Unavoidable sources of error should be borne in mind in determining the relative cardiac dullness. Chief among these are deformities of the bony wall of the thorax (rachitis, bulging of the ribs over the heart, the narrow chest with sharply sloping sides), rigidity of the bony wall, emphysema, large breasts, and the panniculus adiposus of a fat man. The most accurate results are obtained in children and young people, as in them the thorax wall is elastic and the layer of subcutaneous fat usually not thick enough to interfere with percussion.

Serious errors in locating the heart borders are occasionally made by experienced physicians the cause of which is unexplained. When working as a student in Krehl's clinic in Heidelberg, I saw an American physician with cardiac symptoms who had been examined by some of the ablest clinicians in America. No one ever made out any enlargement of the heart, but the orthodiagraph showed that it was markedly enlarged. Even after this discovery we were unable to demonstrate the enlargement by percussion. The patient had a normal shaped chest and there was no evidence of emphysema.

The change from the normal resonance over the lung to a less resonant note over the borders of the heart is not due to absorption of sound waves by the heart, but to the fact that the thinner layer of air-containing pulmonary tissue overlying the heart yields a duller note. (See section on Percussion, page 24.)

In percussing the relative dullness heavy percussion must not be employed, as too strong a stroke will set up vibrations in such a large portion of pulmonary tissue that the heart borders cannot be located. The strength of percussion must also vary with the subject, as the proper stroke depends on the thickness of the chest wall. Here experience is the guide. For the beginner the necessity of varying the force of percussion forms one of the chief difficulties in determining the area of relative cardiac dullness. It can only be overcome by practice.

Technic of the Percussion of the Relative Cardiac Dullness

After the area of absolute cardiac dullness has been determined by the method described in the preceding section (page 291) the right border of relative dullness should be located. The pleximeter-finger should be placed in the fourth right intercostal space at a right angle to the median line. It should be firmly applied to the skin, as deep percussion is desired. The patient should be made to exhale as fully as possible the air from the chest and then to hold his breath. Expiration can be facilitated by pressure of the examiner's hand against the lower part of the sternum. Expiration retracts the margin of the right lung from the heart and this facilitates the location of the right border of the heart. The percussion-stroke should be fairly strong and sustained; that is, the percussing finger should not be brought away quickly from the pleximeter. The first point of definite change from the full lung-resonance to a duller sound should be marked with a skin pencil. If the patient is dyspneic advantage cannot of course be taken of percussing the border with the breath momentarily held in expiration. The border should also be determined in the

interspaces above and below that first selected and the points marked and then connected with a line. The distance of the point on this line farthest from the median line should be measured in centimeters and recorded. This corresponds to the Mr of the orthodiagram; that is, the greatest distance of the right border from the median line and is called the right median distance. (Fig. 28.)

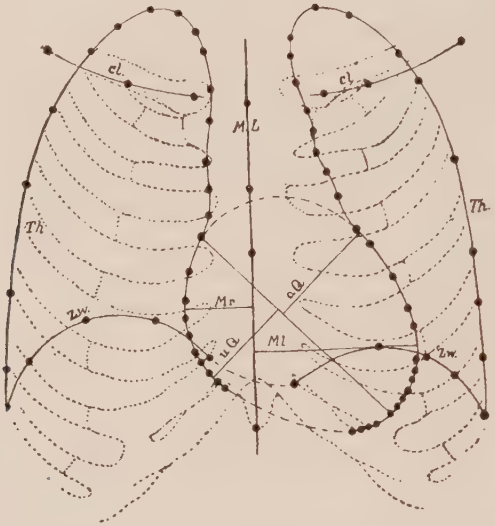


Fig. 28.—Orthodiagram of heart with dorsoventral illumination. Ml median line; Mr distance of right border from median line; Ml distance of left border from median line; uQ + oQ = width (Breite) cl. clavicle; Th inner bony wall of thorax; Zw diaphragm (Seifert and Müller, "Taschenbuch der medizinsch-klinischen Diagnostik," J. F. Bergmann.)

The upper and left borders should then be determined. The subject should breathe quietly. Advantage cannot be taken of withdrawing the lung margins from the heart by expiration because, in expiration, although the position of right border is not changed, the upper and left borders move upward and to the left.

The percussion of the upper border to the left of the sternum should be made with a stronger stroke than that employed for the right border, as both the wall of the thorax and the layer of lung that overlies the heart are thicker here than elsewhere. Percussion of the left border in the region of the apex must be made with a lighter stroke than that employed for determining the other boundaries, because a strong blow will yield a sound of impaired resonance far to the left of the true border of the heart. This portion of the heart is covered with a thinner layer of lung than the other borders of the heart. Hence the necessity of using lighter

percussion. The layer of lung covering the anterior part of the left lateral surface of the heart near the apex is often so thin as to yield a dull sound on strong percussion. If the heart is enlarged, or if the heart is of normal size, but the chest narrow and box-shaped, with the left side beginning near the mammillary line and curving sharply backward, lighter percussion must be employed than when the heart and the shape of the chest are normal. If forcible percussion is used the left border of dullness will correspond to a line far back on the left side of the heart, instead of marking the left border of the anterior surface, the position of which the examiner is trying to locate. The larger the heart or the narrower the chest the greater the error introduced by heavy percussion.

The distance of the point on the left border of relative cardiac dullness farthest from the median line is measured and recorded. This is the left median distance, Ml, of the orthodiagram. The left border is usually just outside the palpable apex impulse, but if the heart is enlarged and beating forcibly, the left border, as demonstrated by the orthodiagram, may be a considerable distance inside the apex-beat. In one of my patients the palpable apex impulse was 11 cm. from the median line, while the left border determined by telefluoroscopy was only 9 cm. from the median line. As a rule, when the heart is producing a forcible impulse over a wide area the left border is in the middle rather than at the left of the area of palpable impulse. The left border is often found to pass through the point of maximum impulse (Moritz).

The under border of the heart should be determined if possible. The method has already been described in the section on absolute cardiac dullness (page 292). The dull tympanitic note over the stomach and left lobe of the liver changes to a dull non-tympanitic sound on percussing upward when the lower border of the heart is reached. In many cases the lower border cannot be made out by percussion, but, in my experience, the location of the left end of this border near the apex can usually be determined, at least approximately. To the right of the left parasternal line, or thereabouts in most instances, the dull note over the liver merges without appreciable change into that over the heart.

Moritz holds that the length of the heart can be made out by percussion, although with less accuracy than the right and left borders. The highest point on the right border of the heart (junction of right auricle with superior vena cava) is joined to the lowest point of the heart, which is usually located below the apex impulse. This distance is measured and compared with the length given in the standard tables of the long diameter of the heart based on orthodiagraphic measurements. If the length of the heart can be determined, even roughly, by percussion it is of distinct value,

for the reason that it is necessary to know the length as well as the transverse diameter in order to judge the size of the heart. If the diaphragm is low the heart assumes a vertical shape, and, even when the heart is enlarged, the distance of the right and left borders from the median line may be within normal limits. The length, however, would be distinctly increased. With a high diaphragm the heart becomes more horizontal in position than normal. The transverse diameter will be found increased, but the length will be normal. The evidence from percussion would indicate dilatation of the heart unless the length was known.

There are weighty objections to the view of Moritz that the "auricle-cava angle," as it is called, can be located by percussion. In the first place, when the patient is standing erect the orthodiagraph shows that often there is no such angle. In a large percentage of the normal persons I have examined the right border of the superior vena cava and the right auricle form almost a straight line. Secondly, I do not believe it is possible to mark out by percussion the width of the great vessels above the heart unless they are so dilated as to extend a considerable distance beyond the borders of the sternum. The reasons for holding that the narrow zone of dullness, normally found on either side of the upper sternum, is not due to the great vessels are well stated by Sahli.[1] Briefly they are as follows: (1) The rigidity of the upper sternum damps vibrations in its neighborhood. This explains in part at least the slight relative dullness just beyond the sternal borders. (2) The thick layers of air-containing tissue forming the anterior lung margins meet in the median line above the third rib. (3) The great vessels pass backwards from the heart as well as upwards. If the position of the right border of the superior vena cava cannot be determined by percussion it follows that its junction with the right auricle cannot be located by this method.

The approximate length of the heart can be ascertained by measuring the distance from the upper margin of relative cardiac dullness in the median line to the apex impulse, or, in case the lower border can be percussed, to the junction of the left and lower borders. This distance according to Sahli corresponds fairly well to the average for normal subjects in Dietlen's tables (see page 395) made up from orthodiagraphic measurements. The vertical diameter of absolute cardiac dullness, that is, the distance from the upper margin of absolute dullness to the level of the lower end of the left border of absolute dullness measured in a vertical line, also serves as a valuable index to the length of the heart.

If the heart is distinctly enlarged to the right there will be marked dullness on rather forcible percussion over the lower part of the sternum,

[1] "Lehrbuch der klinischen Untersuchungsmethoden," sechste Aüflage, Leipzig u. Wien, 1920, Band I, 302.

that is at the level of the costosternal articulations of the fifth ribs, in the median line. It is important for beginners especially to avail themselves of this aid in recognizing dilatation of the right ventricle. Another helpful procedure is to percuss lightly in the median line from above downward. If above the level of the fifth rib, in the median line, the sound becomes absolutely dull, enlargement of the right heart is probable.

If the right border of relative dullness is in the vicinity of the right parasternal line it points strongly to dilatation of the right auricle. Confirmatory evidence of the correctness of this deduction will usually be found in distention of the veins of the neck with visible venous pulsation in the erect position, and, in addition, not infrequently an enlarged pulsating liver (Lewis).

With proper technic the right border of dullness usually agrees fairly well with the right border as found by the orthodiagraph. The left border of dullness will often be found to exceed the orthodiagraphic measurement, and if the chest is narrow, with sharp sloping sides, the error may be great.

The angle formed by the right border of the heart and the upper boundary of the liver is acute, as is shown by the orthodiagraph as well as in anatomical preparations. In the percussion outline the angle is either a right angle or an obtuse one. This is due to the impairment of pulmonary resonance in the neighborhood of the liver by the presence of this airless body in the percussion zone. The heart silhouette obtained by the orthodiagraph often extends above the border of the relative cardiac dullness both to the right and to the left of the sternum. This is explained by the fact that the upper part of the heart is covered by a thick layer of lung-tissue and the presence of the sternum which interferes with percussion.

The results obtained by percussion in cardiac diagnosis must always be regarded as findings which, in every single case, must be subjected to critical scrutiny and correlated with other findings before they can be correctly interpreted. Then, and only then, will one not be disappointed in the percussion of the relative cardiac dullness, even if forced to abandon the idea that it yields results in complete agreement with those obtained by orthodiagraphy (Sahli).

Threshold Percussion of the True Cardiac Dullness
Orthopercussion

The theoretical basis of threshold percussion has already been discussed (see page 22). Goldscheider believes that the vibrations set up by the

lightest percussion-stroke traverse the entire chest. The method has been used chiefly in the attempt to map out the true size of the heart. One should percuss so lightly as to obtain only a slight sound over the lungs. Then the "sound rays" pass unobstructed in their course through pulmonary tissue from the front to the back of the chest. The examination room should be absolutely quiet and the sound emitted by the percussion-stroke should be just loud enough to be heard by the examiner with his ear fairly close to the chest and in front of the point percussed. The sound produced by percussion should be so slight that it is inaudible to others standing near the patient. In passing from the lung to the heart, as soon as the border of the heart is reached, the sound rays passing

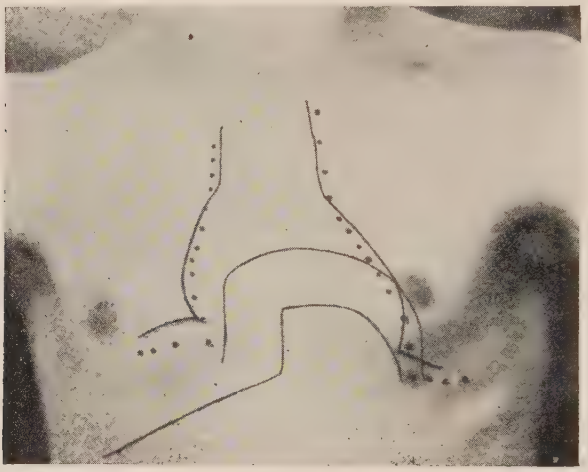


FIG. 29.—Normal absolute and relative cardiac dullness indicated by narrow lines; orthopercussion outline by a broader line; the orthodiagram by the dotted line. (Romberg, "Krankheiten des Herzens und der Blutgefässe," Ferdinand Enke.)

through the chest will be intercepted by the airless structure of the heart and *no sound will be heard:* the relatively thin layer of lung-tissue over the heart not being thick enough to emit a sound. It is essential that percussion should be made over a small area of the skin in a sagittal direction. The manner of percussion devised by Plesch, described on page 21 should be employed. Percussion is made in the interspaces and the points where the faint pulmonary resonance disappears marked with dots by means of a skin pencil. After the percussion of the entire cardiac outline is completed connect the dots with a line. As a valuable check on the results obtained the examiner is advised to repeat the percussion of the heart with his eyes blindfolded.

The value of orthopercussion in determining the true size of the heart is still unsettled. In the hands of Curshmann and Schlayer it yielded excellent results in the cases they selected for examination. Schlayer told the writer that it could not be employed in an ordinary ward or in the examining room of a clinic, owing to disturbing noises. It is a more difficult method for the beginner than the other procedures described in this section for determining the size of the heart. In emphysema and in persons with thick chest walls it certainly cannot always be used successfully. The claim made by its advocates that the outline of the great vessels above the heart can be determined even when not dilated needs more evidence in its support (see page 298). The correctness of the theoretical basis of the method given by Goldscheider is doubtful.

Palpatory Percussion of the Heart (Ebstein's Tast Perkussion)

This depends on the determination of the feeling of resistance. The tissues overlying the heart should offer more resistance on palpation than neighboring portions of the lung that are composed of air-containing tissue of much greater thickness. Percussion is made directly with the finger tips. Ebstein claimed that the cardiac outline could be made out by noting the change in resistance when the borders of the heart were reached. Many experienced physicians believe that a sense of increased resistance is a great and unconscious aid to the examiner in the topographical percussion of the heart by the finger-plexímeter. It has been shown, however, that the importance of palpation in this respect has been overrated. Trustworthy results in outlining the cardiac area cannot be obtained even by skilled examiners when the sense of hearing has been eliminated by having the ears closed.

The Boundaries of the Relative Cardiac Dullness

The outline of relative cardiac dullness is broader below than above. The right and left sides are curved lines that are slightly convex laterally. Its borders are at a variable distance from those of the absolute cardiac dullness. On the right side the relative and absolute dullness are usually separated by more than the width of the sternum; above, near the left sternal border and to the left near the apex, they are nearer together, but even at these points generally 1 to 2 cm. apart. Below, the relative cardiac dullness merges into the liver dullness.

The boundaries are:

Above: third rib. Higher in children. In old age the third interspace.

To the Left: a line slightly convex outwardly within the mammillary line from the upper border to the cardiac impulse or a point just outside the impulse.

To the Right: a finger's breadth outside the right sternal margin. Sometimes at the right sternal margin and rarely at the left sternal margin where it may coincide with the border of absolute cardiac dullness.

It is well not to depend alone on the relation of the heart borders to the anatomical landmarks of the thoracic surface. The mammillary line is too variable in position and the midclavicular line naturally depends on the length of the clavicle which is proportional to the size of the thorax. There is considerable difference also in the width of the sternum in different chests. It is better to express the width of the relative cardiac dullness in centimeters, by recording the distance of the right and left borders from the midsternal line. The normal figures vary with the age, height and sex of the subject (see tables 394). The (Mr), right border of the heart, is 3.5 cm. to 4.5 cm. from the median line. The (Ml), left border is 7.5 cm. to 8 cm. from the median line. The (L), length of the heart as determined by the orthodiagraph can, in my opinion, rarely be made out even approximately by percussion. According to Sahli the distance from the upper border of relative cardiac dullness to the apex corresponds fairly well to the length of the heart—12 to 14 cm.,—given in the standard tables of the cardiac measurements.

Physiological Variations in the Size of the Cardiac Dullness.

Respiration.—In inspiration the expanding borders of the lungs move over the anterior surface of the heart so that the areas of both absolute and relative cardiac dullness are reduced in size. The descent of the diaphragm during inspiration is accompanied by a downward movement of the heart and a shifting of the long axis of the heart toward the vertical line. The upper border of relative cardiac dullness may be found to be 2 to 4 cm. lower at the end of a full inspiration than it was during quiet breathing (Sahli), and the absolute cardiac dullness may be obliterated. In expiration reverse changes occur. The borders of the lungs are retracted from the heart and the upward movement of the diaphragm pushes the heart upward and the apex outward so that the angle which the axis makes with the median line becomes wider and the position of the heart more horizontal. The cardiac dullness is increased above and to the left. The right border is unchanged.

Position.—On changing from the recumbent to the erect posture the diaphragm falls and the heart as shown by the orthodiagram descends and becomes more vertical and somewhat narrower. These alterations in the shape and size of the heart can rarely be made out by percussion. Usually the areas of both absolute and relative cardiac dullness are greater in the erect than in the horizontal position. This is explained by the position of the heart, which lies nearer the anterior wall of the chest when a person is standing. It pushes the lung borders further to each side and this increases the area of absolute cardiac dullness. If the subject leans forward the area of dullness is further increased. It is sometimes found that in cases of emphysema the percussion outline of the heart can only be made out with the individual erect and bending forward. The spine should be kept straight. Percussion in this position is an awkward procedure for the examiner. When the patient lies on the left side the areas of both absolute and relative dullness increase to the left. Lying on the right side increases the dullness to the right. Even when the heart is normal the absolute dullness may then reach beyond the right sternal border and be entirely absent on the left side of the sternum.

The movability of the heart in respiration and with change in position varies greatly with different individuals.

Age.—The area of dullness varies with age. In children the diaphragm is higher and in old age lower than in early adult life. The heart in childhood as a result of the high position of the diaphragm is higher in the chest and more horizontal and extends further to the left. The heart in old age on the other hand is lower, more vertical, and extends less far to the left.

Pathological Changes in the Size of Cardiac Dullness

1. Increase in the Area of Cardiac Dullness Due to Myocardial Disease.—The most common cause is dilatation of the left or right ventricle or of both ventricles. Less frequently increased dullness is due to dilatation of the left auricle or of the right auricle or to the presence of fluid in the pericardial cavity (pericardial effusion). Hypertrophy of the ventricle unaccompanied by dilatation rarely produces an increase in the area of dullness. Personally I have never seen an undoubted instance of definite increase in cardiac dullness that was found at autopsy to be due to hypertrophy alone. Hypertrophy of the ventricular wall would have to be of high degree in order to increase the area of dullness. An increase of 5 mm. above the normal thickness of the ventricular wall is consider-

able and yet it is admitted by Moritz and others that an increase in the size of the heart of 5 mm. or less at the right or left border cannot be detected by percussion. If 5 mm. at one border is within the limit of error of the method, then an increase of 5 mm. at both right and left borders, or 1 cm. in the total width of the heart, would have to be exceeded in order to increase the width of the cardiac dullness.

Increase in the size of the left or right ventricle increases both the areas of absolute and relative dullness to the left. (Fig. 30.) It is

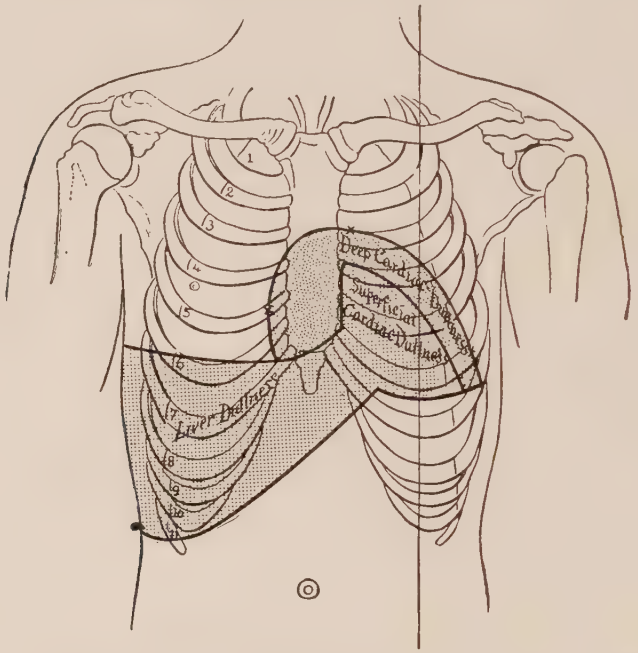


Fig. 30.—Cardiac dullness in dilatation of the left ventricle. (Sahli, "Diagnostic Methods," W. B. Saunders Company.)

important for the student at the outset of his study of cardiac percussion to grasp this fact. Often both student and practitioner have the false idea firmly fixed that in dilatation of the right ventricle the dullness is increased to the right and this change in the area of dullness differs from that in dilatation of the left ventricle, in which the area of dullness is increased to the left. The anterior surface of the heart is formed by the right auricle and the right ventricle except a thin rim of left ventricle which forms the left border. When the right ventricle enlarges it pushes the left ventricle to the left and backward. The right ventricle is increased in its transverse and also in its vertical and in its anteroposterior diameters. It rarely displaces the right border of the heart to the right

a sufficient distance to be definitely determined by percussion of the relative cardiac dullness. Dilatation of the right ventricle does, however, often push the medial border of the right lung further to the right and this increases the area of absolute dullness to the right and gives the step-like contour of the right border of absolute dullness described by Krönig. An increase of the absolute dullness beyond the right border of the sternum without any change in the position of the right border of the heart as revealed by the orthodiagraph or of the right border of relative dullness, is usually an indication of dilatation of the right ventricle. The increase in the anteroposterior diameter of the right ventricle due to dilatation of its cavity gives rise to an increased dullness over the lower sternum in the median line. This is a fact of considerable diagnostic importance, as I have found in my own experience. In dilatation of the right ventricle both the relative and absolute dullness are increased upwardly to the left of the sternum. Relative dullness often begins as high as the second rib and absolute dullness at the third rib.

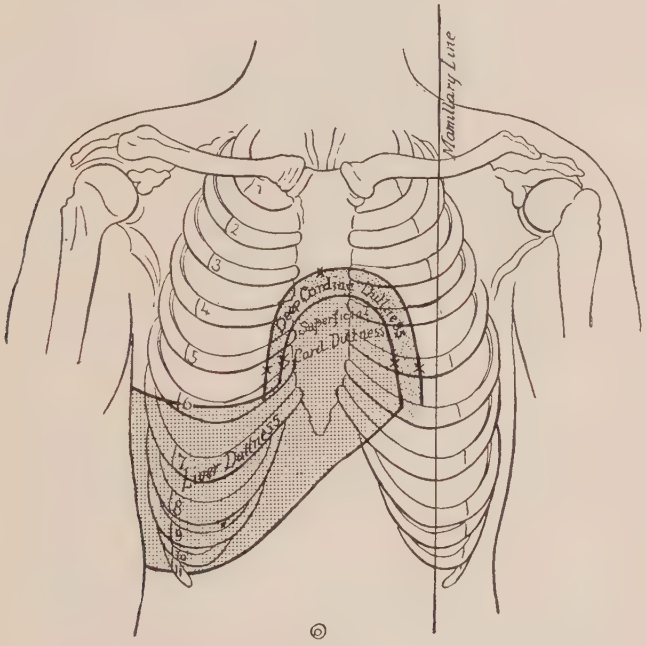


Fig. 31.—Cardiac dullness in dilatation of the right heart (ventricle and auricle). (Sahli, "Diagnostic Methods," W. B. Saunders Company.)

In dilatation of the left ventricle the dullness is increased to the left and below. A marked increase in the area of dullness in an upward direc-

tion is a characteristic feature in dilatation of the right ventricle while an enlargement downward is a sign of dilatation of the left ventricle.

The only portion of the left auricle that presents anteriorly is the appendix. In mitral stenosis the auricular appendix may be so greatly dilated that it causes an increase of the absolute cardial dullness, the outer part of the upper border near its junction with the left border being carried upward to or above the third rib and outward toward the parasternal line or even beyond it.

If the right auricle is greatly dilated the contour of the right border of absolute dullness may become convex outwardly forming an acute angle below at the junction with the liver flatness (Sahli). (Fig. 32.)

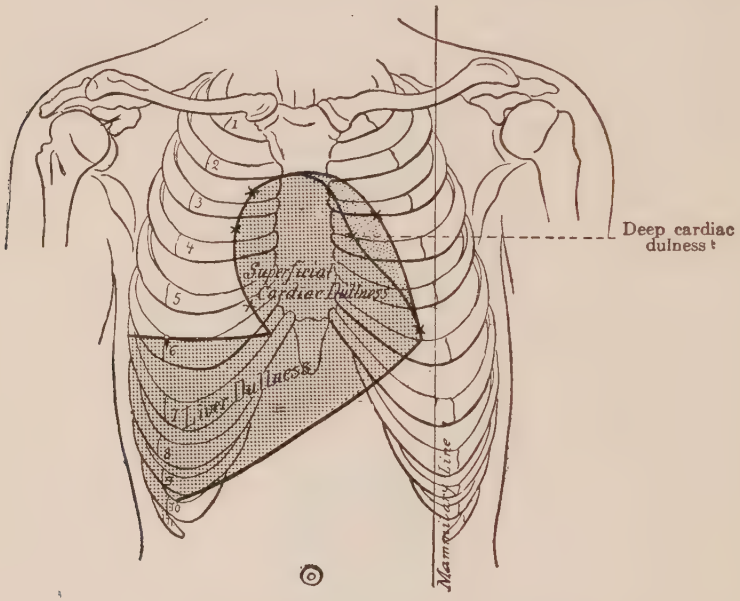


FIG. 32.—Dilatation of the right auricle and ventricle in a case of tricuspid insufficiency. The border of absolute cardiac dullness coincides with that of the relative except on the left side. (Sahli, "Diagnostic Methods," W. B. Saunders Company.)

Hypertrophy and dilatation of the left ventricle occur in aortic insufficiency, aortic stenosis, mitral insufficiency, chronic interstitial nephritis, primary hypertonia, and cardiosclerosis.

Hypertrophy and dilatation of the right ventricle occur in mitral stenosis, mitral insufficiency, pulmonary stenosis (rare), tricuspid insufficiency and cardiosclerosis.

Hypertrophy and dilatation of the left auricle occur in mitral stenosis and insufficiency.

Hypertrophy and dilatation of the right auricle occur in tricuspid stenosis and insufficiency. The latter condition is usually secondary to failure of both left and right ventricles. (Fig. 33.)

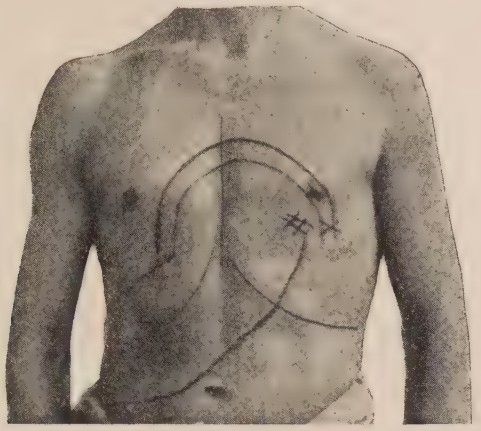


FIG. 33.—Mitral stenosis and regurgitation with cardiac insufficiency. Congestion dilatation of the right auricle; chronic passive congestion of the liver. X apex-beat of the left ventricle; # left border of right ventricle, from here to middle of sternum, diffuse heaving pulsation. (Romberg, "Krankheiten des Herzens und der Blutgefässe," Ferdinand Enke.)

2. **Increase in the Area of Cardiac Dullness due to Pericardial Effusion.**—Fluid first accumulates at the base of the heart. If the patient is recumbent in bed when the fluid develops, flatness may first be detected in the neighborhood of the attachments of the third costal cartilages to the sternum. Usually abnormal dullness extends to the right of the sternum and below the apex-beat. In some cases at an early stage dullness may be elicited in the fifth right intercostal space (Rotch's sign). The area of absolute dullness increases in all directions as the distending pericardial sac pushes back the lung borders including the lappet of the left lung that embraces the apex of the heart. On the right side the absolute dullness extends out in the fourth and fifth interspaces sometimes reaching as far as the right nipple line; on the left it may extend to the anterior axillary line, or if the effusion be very large even to the midaxillary line. Above to the left of the sternum the flatness may reach the second interspace. The left border of dullness in pericardial effusion is much further beyond the visible and palpable impulse than in health or in cases of dilatation of the heart. This is a valuable aid in distinguishing pericardial effusion from cardiac dilatation. The area of absolute dullness gradually approaches to that of the relative dullness

so that the lines marking the two areas may lie close together or even coincide.

The angle between the upper level of liver flatness and the cardiac flatness, the cardiohepatic angle, becomes obtuse. This is an undoubted and frequent finding in cases of pericardial effusion, in spite of the fact that the radiograms often show that the angle between the heart and liver, even when the effusion is great, is either an acute or a right angle. The explanation of the wide angle obtained on percussion is simple to anyone familiar with the acoustics of percussion. Vibrations produced by striking a point on the chest spread not only centrally in the direction of the blow, but in all directions. Solid as well as air-containing tissues in the zone of vibration influence the resulting sound. Even the effect of a light percussion-stroke is damped when the place struck is near the liver and heart by the presence of these airless bodies in the zone of vibration. As a result what is seen to be a narrow acute angle with the x-ray may become an obtuse angle in the percussion outline of the right border of the heart at its junction with the liver. An obtuse angle is also obtained in some cases of dilatation of the right heart and may be caused by an excess of fatty tissue in the mediastinum in front of the heart. In cases of suspected pericardial effusion the cardiohepatic angle should be marked on the skin and percussion repeated from day to day. A rapid increase in the width of the angle would favor the diagnosis.

The pericardial sac is much wider below than above, hence when distended with fluid the area of cardiac dullness is much wider below than above. The outline of dullness often assumes a pyriform shape with the stalk of the pear above. Sometimes the outline is nearly that of an equilateral triangle with the apex near the middle of the sternum usually at the level of the second ribs. (Fig. 34.) The downward curve to the left may show an indentation at or about the junction of the upper and middle third of this border of dullness (Sibson's notch), and the narrow upper portion of dullness a peak. The fluid is lighter than the heart and if the effusion be considerable the area of dullness is usually greater in the erect than in the recumbent position.

The characteristic form of the percussion outline will be modified if there is emphysema or if the lung borders are adherent. The size and shape of the area of dullness will depend upon the size of the exudate, and differ so in different cases that the diagnosis of pericardial effusion must be based on additional evidence to that furnished by percussion. In some cases of great dilatation of the heart the area of dullness is very similar to that seen in pericardial effusion.

Small effusions cannot be detected by percussion. Probably the

amount must be more than 300 cc. as Morris and Little found the presence of fluid could not be recognized by the Roentgen ray until 250 cc. or 300 cc. were injected into the pericardial sac. The study was made on bodies soon after death.

Skodaic resonance may be present over the pulmonary tissue beyond the borders of cardiac dullness in pericardial effusion due to compression of the lungs by the distended pericardial sac.

3. **Decrease in the Area of Cardiac Dullness.**—This is never due to diminution in the size of the heart. Atrophy of the heart occurs. It was found (brown atrophy) often in cases of malnutrition during the war, but only at the autopsy table. It could not be recognized on physical ex-

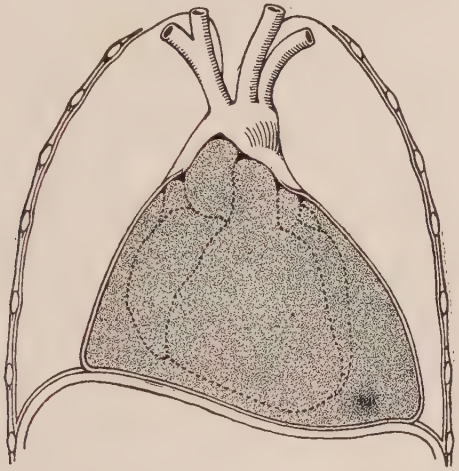


FIG. 34.—Effusion into pericardium (pericarditis exsudativa). Typical triangular area of dullness on percussion with rounded apex of triangle above. (Külbs, "Leitfaden der medizinischen-klinischen Propädeutik," J. Springer.)

amination, as the reduction in size of the heart was not sufficent to affect the area of dulness.

Pulmonary emphysema is the most frequent cause of diminution of the area of cardiac dullness. The area of absolute dullness frequently disappears, and the outline of relative cardiac dullness obtained is usually quite an untrustworthy guide to the size of the heart. If emphysema exists cardiac dilatation of high degree may be overlooked.

Meteorismus (filling of the stomach and intestine with air) may render cardiac percussion not only valueless but misleading. Distention of the stomach and intestine with air presses up the dome of the diaphragm and imparts a tympanitic quality to the sound over the heart even when light percussion is used. In a case of meteorismus the outline of absolute

cardiac dullness, as I made it out, was normal, yet at the autopsy the following day the heart weighed over 600 grams and both the right and the left ventricle were markedly dilated.

In *pneumopericardium* (air in the pericardial sac) the dullness disappears and is replaced by a tympanitic or metallic sound. This area of tympany shifts when the position of the subject is changed. Pneumopericardium is a very rare condition. In one of my cases of left sided pneumothorax without pneumopericardium (diagnosis made at autopsy), there was obliteration of the relative and absolute cardiac dullness. In another very rare condition, mediastinal emphysema (entrance of air into the mediastinum), if the air accumulates in considerable amount in the anterior mediastinum the cardiac dullness becomes obliterated. The area of tympany does not move with change of posture.

Displacement of the Heart

This has already been considered in its relation to pulmonary disease (page 124), and also in connection with displacement of the apex-beat. The normal position of the heart in the chest is dependent on the position of the diaphragm and of the mediastinum. Dislocation of the heart results when the position of either the diaphragm or the mediastium changes. The mediastinum is held in a position of equilibrium determined by the difference of pressure in the two pleural cavities. When the negative pressure in one pleural cavity becomes less or changes to a positive pressure, the mediastinal wall, and with it the heart, moves toward the side in which the absolute pressure is less.

Displacement of the apex impulse to the left is not sufficient to distinguish displacement of the entire heart from dilatation of the organ. It is necessary to supplement palpation with percussion of the right border, as in dislocation of the heart the transverse diameter is of course not increased. If the right border is further to the right than normal it is necessary to percuss out the left border to determine whether the condition is dilatation or displacement of the heart.

Displacement downward of the area of cardiac dullness is most commonly caused by emphysema of the lungs.

Lateral displacement is due to increase of pressure in one pleural cavity. This occurs in pneumothorax, pleural exudate, tumors of the lung and mediastinum. In these pathological processes the displacement is away from the diseased side of the thorax because the pressure on that side is increased. Lateral displacement is also due to decrease in pressure. In

contraction of the lung due to atelectasis, fibroid phthisis, or chronic pneumonia, the pressure on the diseased side is diminished and the heart is pushed toward the side of the lesion. The upper level of cardiac dullness is higher than normal as the heart is pushed upward as well as to the side. The absolute dullness is usually increased as the lung borders are drawn back from the surface of the heart.

The area of cardiac dullness frequently merges with that of the pleural exudate or tumor so that it is impossible to determine one or more borders of the heart by percussion.

Upward displacement of the cardiac dullness is due to ascites, pregnancy or abdominal distension with gas.

Deformities of the thorax, especially kyphoscoliosis, displace the area of cardiac dullness variously and render percussion difficult and the results misleading.

In situs inversus and dextrocardia without displacement of other viscera, which is even more rare, the cardiac dullness is on the right side forming a mirror-picture of the normal form and position.

When the heart is displaced to one side by a pleural exudate or pneumothorax the borders of the lung are pushed away from the heart so that the absolute dullness increases and approaches that of the relative cardiac dullness. In fact the areas of relative and absolute dullness may coincide. Also when the heart is pushed upward by abdominal distention the area of cardiac dullness becomes not only higher but wider. This is in part due to retraction of the borders of the lung. Rise of the diaphragm diminishes the negative intrathoracic pressure and this causes a retraction of each lung toward its hilus. The upward movement of the diaphragm pushes the apex of the heart, which rests upon the diaphragm, outward and to the left, while the position of the base of the heart is changed but little. This pendulum movement of the heart to the left increases the width of the cardiac dullness.

Dullness in the Region of the Great Vessels

Normally there is resonance over the upper part of the sternum on fairly light or strong percussion. The rim of dullness on either side of the sternum is probably due to the proximity of the sternum and not to the great vessels which lie far below the surface of the chest at the level of the first and second ribs and are covered by a thick layer of pulmonary tissue. In mediastinal tumors, persistent thymus, substernal goitre, in syphilitic aortitis and in dilatation of the aorta in arteriosclerosis, there

is often dullness over the manubrium which may be relative or absolute. It is usually separated from the area of absolute cardiac dullness below. In aneurism of the aorta there is often a wide area of dullness above the third rib. It is present over the sternum and extends beyond one or both of its lateral borders. In aneurism of the ascending portion of the aorta the dullness is to the right of the sternum, while in aneurism of the arch of the aorta it tends to extend beyond the left sternal border. In the congenital disease, patent ductus arteriosus, there is sometimes a rim of absolute dullness to the left of the sternum, due to dilatation of the pulmonary artery, from the level of the second to the fourth costal cartilage. Below, this area often merges with that of the absolute cardiac dullness.

AUSCULTATION OF THE HEART

Introduction

In the minds of most students and practitioners auscultation of the heart is regarded as the most important means of ascertaining whether the heart is normal or diseased. Careful history-taking, inspection, palpation and percussion are apt to be neglected or even omitted, but never auscultation. This is a serious mistake. The great Laennec recognized that abnormal heart-sounds—bruits or murmurs (see page 316)—could occur in the absence of valvular disease and he tended to minimize the importance of auscultation in the study of valvular disease of the heart. His immediate successors, however, seized upon the method, and its great value in diagnosis was quickly proved. Within the last twenty years it has been for the first time generally recognized by medical teachers that auscultation has often led to serious errors in diagnosis. For it has been discovered that murmurs previously regarded as definite evidence of disease are frequently heard over the normal heart. To Sir James Mackenzie credit is chiefly due for pointing out clearly the error of expecting to gain from auscultation information regarding the condition of the heart that auscultation cannot yield.

On examining the heart the chief thing to be determined is the strength of the organ and not its anatomical condition. Students must learn to think physiologically if they are to discover the cause of the symptoms of which patients complain. They are dealing primarily with disturbances of function rather than with alterations in structure. More can be learned of the ability of the heart to do its work from a good case history and from simple functional tests than from auscultation.

Auscultation of the heart is an interesting study. To eager students it is really fascinating. Proficiency in auscultation is acquired more easily than proficiency in percussion. The results obtained by percussion are easily controlled by radiography, while there is no similar way of checking up the findings on auscultation. It is true that something has been learned by the graphic registration of the heart-sounds and the work of Einthoven and others promises much in the future from the development of phonocardiographic study. The results obtained by auscultation of the heart must always be carefully interpreted and compared with the other findings or they may mislead the observer. Considered alone and as sufficient in themselves they have so frequently led to error that Mackenzie has gone so far as to maintain that progress in the clinical knowledge of heart disease was hindered rather than advanced by the discovery of the stethoscope.

The Heart-Sounds

On listening over the cardiac area two sounds are heard with each beat of the heart. One sound accompanies the impulse, one sound follows it. The former is called the first or systolic sound, the latter the second or diastolic. They are most closely imitated by pronouncing the two syllables, lubb dupp. The two heart-sounds are separated by a short pause and the two sounds produced by a single heartbeat are followed by a longer pause. The first sound begins at the onset of systole and is heard throughout nearly the first half of systole. The second sound begins and ends in the early part of diastole. Systole is shorter than diastole, hence the first sound follows the long pause. The first sound is loudest over the fourth or fifth interspace just within the left nipple line. In this region the accent is on the first of the two syllables, thus—lubb′ dupp, lubb′ dupp,—while at the base of the heart the accent is on the second syllable—lubb dupp′, lubb dupp′. At the apex the first sound is the louder at the base the second sound. The first heart-sound is normally duller and of lower pitch and of longer duration than the second sound, which is sharper.

It is important to recognize which sound is the first and which the second. There are four aids to proper timing of the sounds. First: distinguish the long from the short pause. This is easy unless the heart rate is rapid. The first sound is preceded by the long pause. Second: the accent at the apex is on the first sound. Third: the character of the first sound is different from that of the second. Fourth: the first sound is longer and louder at the apex and ends less abruptly than the

second sound. The first sound coincides with the outward systolic thrust of the apex. It marks the beginning of systole. If the impulse can be felt one should note with a finger placed beside the bell of the stethoscope the sound which occurs at the same instant as the thrust. This is the first sound. It may be easier in some instances to recognize the sound that accompanies the apex-beat by transdigital auscultation (Riesman). For this procedure a diaphragm stethoscope is used. The tip of the finger flexed at a right angle is placed over the apex-beat. The disc of the stethoscope is then laid on the horizontal phalanx of the flexed finger. It is obvious that this combination of palpation and auscultation is a distinct aid in recognizing the sound that occurs synchronously with the elevation of the finger. In diseased conditions it may sometimes be impossible to distinguish between the first and second sound, because the guides to their recognition can no longer be utilized owing to altered conditions. As the heart rate increases the long pause shortens, until the ear of the examiner may be unable to distinguish it from the short pause between the two sounds. The accent on the first sound may be missing, and the character of the two sounds may become similar. If these deviations from the normal occur and the cardiac impulse cannot be felt, it is evident that it will be impossible to distinguish between the first and second sound.

Third Heart-Sound.—Early in diastole a faint sound resembling a feeble echo of the second sound is sometimes audible near the apex, especially in young people. Often it may be made out only when the person is lying on the left side. Thayer heard it in about 30 per cent of the normal subjects he examined. It is most frequently present between the age of 10 and 20 (84 per cent according to Thayer) and never in old people. It is much fainter than the first sound (according to Einthoven 200 times softer). Very few clinicians recognize the presence of this sound with anything like the frequency indicated by Thayer's statistics and the phonocardiographic records (Einthoven) make it seem probable that it is less common. Thayer has a very acute ear and is a highly trained auscultator. It is not surprising that he should be able to detect this faint sound when listening carefully for it when its presence would have been unrecognized by many good observers.

The Valvular Areas on the Chest

Sounds or murmurs produced at the different valvular orifices are not always heard loudest on the surface directly over the valves. They are

transmitted with varying degrees of intensity to different parts of the anterior wall of the chest. This depends (1) on the conducting power of the tissues between a valvular orifice and the surface of the chest, and (2) on the direction of the blood-current.

The valves lie close together near the junction of the third left costal cartilage with the sternum but at different levels. (Fig. 35.) The pul-

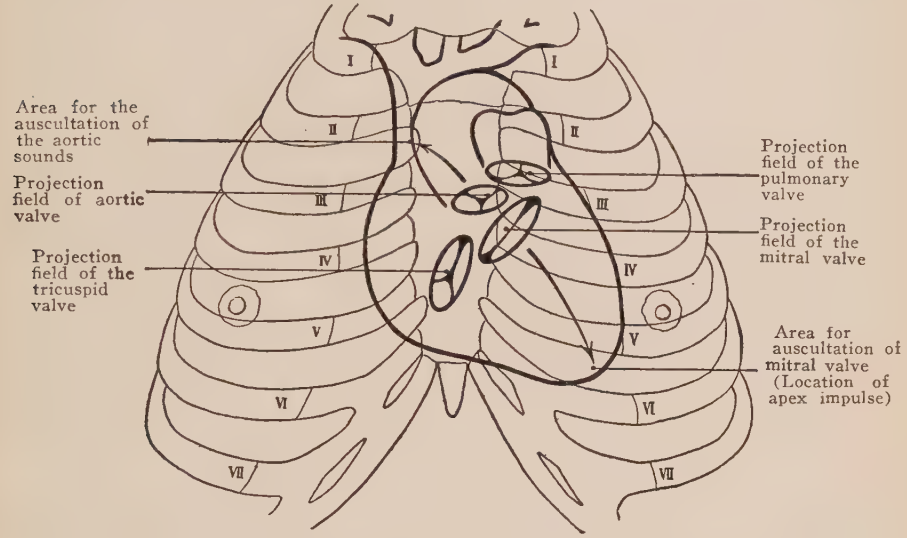


FIG. 35.—Projection of valvular orifices of the heart on the anterior chest wall. (Corning, "Lehrbuch der topographischen Anatomie," J. F. Bergmann.)

monary orifice lies nearest the surface and sound produced here is heard with maximum intensity directly in front of the valve in the second left interspace close to the sternum. This is called the pulmonary area. Murmurs produced at the other three orifices are best heard some distance from their anatomical location. The aortic valve lies deeper in the chest than the pulmonary valve and behind the sternum. The sound is transmitted from the aorta to the chest wall and is usually heard loudest in the second right interspace because the first portion of the aorta approaches the surface of the chest. This is known as the aortic area. The aorta just above the valve is almost in contact with the sternum and for this reason diastolic murmurs are conducted down the sternum and along its left border. Sometimes they are louder at the xiphoid cartilage than at the aortic area.

Occasionally sounds produced at the aortic orifice are best heard directly in front of the valve over the sternum at the level of the third ribs. The mitral orifice lies more deeply in the chest than the other valves, and is

on a level with the upper border of the third left cartilage behind the edge of the sternum. Mitral murmurs are usually loudest where the left ventricle becomes most superficial in the region of the apex. They are heard with maximum intensity in the fifth interspace between the parasternal and nipple lines. From the tricuspid valve sounds are transmitted through the right ventricle and the point at which they are heard with maximum intensity is either the fifth right interspace near the sternum or over the lower end of the sternum.

Mediate and Intermediate Auscultation

Direct auscultation is much less valuable in the study of the heart than indirect auscultation with a stethoscope. This is due to the importance of listening over small areas, especially to the so-called valvular areas just described. The location and maximum intensity of sounds and murmurs can be much better determined by means of the bell of the stethoscope than by the unaided ear. Direct auscultation is of value, however, in detecting some high-pitched tones such as those of diastolic murmurs produced at the aortic orifice. The transmission of systolic mitral murmurs toward the apex is largely through the papillary muscles. The direction of conduction is the reverse of that of the regurgitant blood-current. The presence of the murmur in the left axilla is chiefly due to the proximity of the anterior papillary muscle. The transplantation of a papillary muscle alters the direction in which the murmur is conducted. The statement is often made that murmurs of mitral insufficiency are sometimes conveyed by regurgitation of blood into the left auricular appendix and hence are heard loudest over the third left cartilage or the second left intercostal space (pulmonary area). Most of the systolic murmurs, however, heard with maximum intensity over the pulmonary area do not arise at the mitral orifice but at the pulmonary orifice.

Nature of Heart-Sounds and Murmurs

Two different types of sound are produced by the heart. These are (1) the normal heart-sounds and (2) murmurs. The former are called in English sounds and in German tones, although they are not simple sounds and bear no relation to a musical note. They are in a physical sense noises and so are murmurs. That is, both sounds and murmurs are made up of vibrations, which differ from one another in time and ampli-

tude. A true musical note is made up of vibrations occuring at a definite rate per minute. Pitch is determined by the frequency of the vibrations. The heart-sounds are vibrations set in motion by a force that acts but once and then ceases. In other words the structures whose vibrations produce the sound are affected by a single disturbance of the equilibrium. It is comparable to the sound made by a single blow on a drum. The amplitude of the vibrations steadily decreases from the beginning of the sound (see Fig. 36). Murmurs are due to a force that makes a series of impacts on the vibrating substances. The result is that the initial amplitude is maintained at the same height for a time before it begins to decrease (see Fig. 37). The murmur, with its repeated disturbances of the equilibrium of the vibrating parts, may be compared to the sound produced by passing the violin bow across a string of the violin; the heart-sound, with its single change of equilibrium, to the sound which arises when a violin string is picked with a finger.

Both heart-sounds and murmurs differ in pitch, loudness and duration. Heart-sounds seem to the ear to begin more suddenly and to end more suddenly. Murmurs are apt to fluctuate in intensity. They are usually

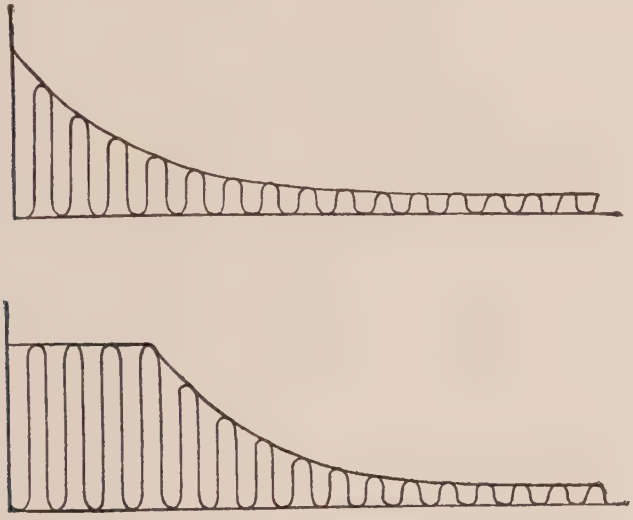


Fig. 36.—The dying away of the vibrations of a sound.
Fig. 37.—The dying away of the vibrations of a murmur. (Geigel, "Lehrbuch der Herzskrankheiten," J. F. Bergmann.)

of longer duration than heart-sounds. The short duration of the heart-sounds and the longer duration of the murmurs which more or less fills the pauses between the heart-sounds is clearly demonstrated in phono-

cardiograms made by Einthoven's method. The heart-sounds are received by a microphone in which they produce variations in the electrical current, as in a telephone receiver, and these electrical changes are registered by a string galvanometer and photographed. The curves do not record the sound vibrations but the electrical changes in potential produced by them. They do, however, permit the accurate timing of heart-sounds and murmurs.

Murmurs may at times be heard over a healthy heart and hence their presence is by no means an indication of disease of the heart, as is often thought.

Sometimes a heart-sound has a murmurish quality. This is usually due to the presence of an indistinct murmur in addition to the heart-sound.

Cause of the Heart-Sounds

The first sound occurs during the first part of systole. It marks, as already stated, the beginning of systole. It occupies the isometric period (see page 250). At this time the ventricle is a closed cavity, as both the auriculoventricular and the aortic valves are tightly shut. There is undoubtedly a valvular element and a muscular element in this sound. The vibrations of the auriculoventricular valves produce part of the sound and the vibrations of the muscular wall of the ventricle another part. The higher the intraventricular pressure the louder the sound. (Wiggers). The sudden distention of the aorta with the blood from the ventricle also sets up vibrations in the walls of the former, which give a third and later element to the first heart-sound. This occurs after the isometric period, which lasts .06 to .07 of a second. Hence there are three elements in the first sound, (1) the valvular, (2) the muscular, and (3) the arterial. In addition there is a definite muscle sound produced by forcible contraction of the auricle. This can be distinctly heard in some cases of heart-block (Cohn). Some authors include the auricular element as a part of the normal first sound.

The second sound occurs at the beginning of diastole. (Fig. 38.) The arterial valves close noiselessly, as the sound begins appreciably later than the sudden diastolic drop in intraventricular pressure, and hence after the valves have closed (Wiggers). The pressure at the beginning of diastole is high in the aorta and relatively high in the pulmonary artery, but the relaxing muscular walls of the heart reduce it to zero in the ventricle. This great difference in pressure sets up vibrations in

the semilunar valves immediately after their closure which give rise to the second sound.

There is some doubt as to the cause of the third sound. It may be due to the sudden tension of the auriculoventricular valves resulting from the first inrush of blood from the auricle to the ventricle in early diastole (Hirschfelder, Thayer). Henderson holds that at the end of rapid filling of the heart, which occurs early in diastole, the auriculoventricular valves are closed by the elastic recoil of the ventricular walls. A wave in the venous pulse called the *h* wave has been found by Hirschfelder, Gibson and Thayer, to occur at the same time in diastole that the third sound is heard. Einthoven thinks the third sound is due to after-

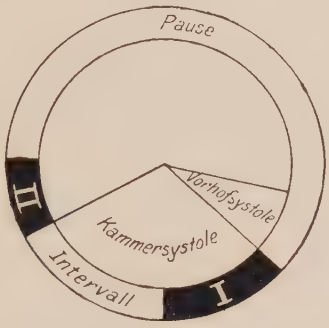


FIG. 38.—Diagram of the cardiac cycle—average heart rate, 72 beats in a minute. I occurrence and length of first sound in early ventricular systole; II occurrence and length of second sound in early ventricular diastole. Note that the pause between the heart-sounds (ventricular diastole) is much longer than the ventricular systole. (Gerhartz, Ztschr. f. klin. Med. Berl.)

vibrations of the aortic valve. Lewis heard the sound in one case during auricular systole at a time when the auriculoventricular valves must have been open.

Intensity of the Sounds

The loudness of the sounds varies in health. Much depends on the amount of material between the heart and the surface of the chest. In fat people, in pulmonary emphysema and in pericardial effusion the heart sounds are weaker, for the distance between the heart and the ear is increased. In the recumbent position the heart falls back somewhat from the anterior chest wall and hence the heart-sounds are fainter than in

the upright posture. Whatever weakens the force of the contraction of the heart may weaken the heart-sounds, especially the first sound. Hence in cardiac weakness due to disease of the heart muscles the sounds may be weak. In cases of defective intraventricular conduction, the first sound is apt to be weak (Oppenheimer and Rothschild). If the heart rate is slow or the diastolic blood-pressure low, the first sound at the apex may be feeble (Wilson), and it is often enfeebled in severe cases of angina pectoris. In many cases, however, of severe cardiac insufficiency the heart-sounds are well heard. The strength of the radial pulse is often a better index to the condition of the heart than the loudness of the heart-sounds. A far better guide than either is the patient's response to exertion. If he can move about freely without dyspnea or pain the heart muscle must be fairly strong. Weakness of the second sound is less common than of the first. The second sound may be weakened, especially in the acute infectious diseases such as pneumonia and typhoid fever, and its enfeeblement is an ominous sign. If only one sound is heard at the apex, it is usually the second sound that is absent. If only one sound is present at the base, it is generally the first sound that is missing.

Accentuation of the Second Sound.—Except in childhood the second aortic sound is louder than the second pulmonic sound. When there is definite increase in loudness of either sound it is called accentuated. The term is applied to an absolute increase and not to relative increase compared with the other second sound. For one sound might be deficient in loudness, the other second sound remaining natural. An increase of blood-pressure in either the general or pulmonary circulation usually increases the loudness of the second sound. The blood-pressure in the aorta is much higher than in the pulmonary artery and the reason that the second pulmonic sound is as loud or louder than the second aortic sound is that the pulmonary valve is much nearer the front of the chest than the aortic valve.

Accentuation of the Aortic Second Sound. This occurs in disease states in which the blood-pressure is elevated, namely, primary hypertonia and chronic nephritis. It can be demonstrated in about two-thirds of the cases of arterial hypertension (Norris). Its absence is sometimes due to weakness of the heart muscle (cardiac insufficiency) and possibly to emphysema and obesity. Many cases are seen, however, in which none of these conditions are present and yet the aortic second sound is not accentuated in spite of a high systemic blood-pressure. In sclerosis of the ascending portion of the thoracic aorta even when the

blood-pressure is normal the second sound is unusually loud and has a ringing quality. In syphilitic aortitis there is often a bell-like ringing accentuation that is characteristic of this disease. After severe physical exertion and in simple nervousness there is often transitory accentuation of the aortic second sound.

Accentuation of the Pulmonary Second Sound. Up to adult age the pulmonary second sound is louder than the aortic second sound. When the sounds are of nearly the same loudness it is impossible to tell which is louder. Geigel found that one second sound must be twice as loud as the other to be recognized as definitely accentuated. Hence the well-known observations of Creighton have little value. She studied 1000 unselected subjects and attempted to distinguish in each person whether the aortic or pulmonic second was the louder. Bock used a differential stethoscope in the study of the intensity of the heart-sounds, but this method is also subject to criticism (Geigel.)

The middle of the pulmonary orifice is one-half cm. further to the left and in front of that of the aorta. If the stethoscope is placed at a point 5 cm. to the right or left of the middle of the sternum the distance from one arterial orifice will be 5 cm. and from the other 6 cm. in the second interspace. The ratio of the intensity of each of the two sounds will be as 25 : 36. That is, if one listens 5 cm. to the right of the mid-line, the intensity of the aortic component of the sound is indicated by the figure 36, the pulmonic component by the lower figure 25. But if one listens closer to the source of sound, namely at the edge of the sternum, the distance will be reduced to 1 and 2 cm. The mixture of the two components is then 1 : 4. Hence, at the right sternal border the intensity of the aortic element is four times that of the pulmonary (Geigel). One sound can be definitely recognized as louder than another when its intensity exceeds that of the weaker sound in the ratio 4 : 3 (Renz and Wolf). As the second aortic and pulmonic sounds are never heard alone but always mixed with the other second sound, a difference in loudness can be only clearly recognized when one sound is at least twice as intense as the other. The bell of the stethoscope should be placed close to the edge of the sternum, first in the second interspace on one side of the sternum and then on the opposite side.

Accentuation of the second pulmonic sound occurs in mitral stenosis and mitral insufficiency due to increased pressure in the pulmonary artery. It is a valuable sign of hypertrophy of the right ventricle. If the right ventricle weakens, the blood-pressure in the pulmonary artery falls and the second sound becomes weaker.

Reduplication of the Heart-Sounds

Doubling of a heart-sound sometimes occurs. (1) The doubling may be incomplete, that is, the second part of the double sound begins before the first is ended. The sound is partly broken in two. This is called a *splitting* of the sound. (2) The doubling may be complete. The two portions of the sound are separated by a distinct interval, so that two sounds are heard instead of one. This is called reduplication of the sound. Reduplication of the first sound gives an anapestic rhythm. ⏑⏑— The accent is on the last sound (lubb-lubb-dúpp). Reduplication of the second sounds yields a dactylic rhythm. —⏑⏑ The accent is on the first of the three sounds (lúbb-dupp-dupp).

Reduplication of the first sound at the apex is common in health. It is intermittent and closely related to respiratory movements. The first sound is doubled at the end of expiration and beginning of inspiration. Doubling of the first sound is more frequent in mitral stenosis than in any other disease. The accent is usually on the second systolic sound. In some cases the first portion of the reduplication may be due to the contraction of the hypertrophied auricle. This occurs in cases in which the interval between the auricular and ventricular systole is increased. The contraction of the auricle has been shown definitely in some cases of heart-block to produce a sound.

The first sound is composed of several elements. One of these, the arterial sound, occurs late in systole, during the expulsion of blood from the heart. This sound usually merges with the other elements to produce a single auditory impression, but if the isometric period is prolonged an interval of silence may precede the arterial sound causing a reduplication (Geigel, Sahli). Reduplication of the first sound has been associated with a block of the right branch of the bundle of His.

Reduplication of the second sound. This is less common than doubling of the first sound. It occurs especially in mitral disease, is most marked at the pulmonary area and is associated with increase of blood-pressure in the pulmonary circulation as revealed by an accentuated pulmonary second sound. It is attributed to asynchronous closure of the aortic and pulmonic valves. Reduplication of the second sound is often noted at the apex in mitral stenosis.

Pendulum Rhythm. Embryocardia

It has been maintained that even with a normal heart rate the pause between the systolic and diastolic sound may be so lengthened as to equal

the pause between the diastole and the succeeding systole (Pawinski).[2] The sound is similar to the tick-tack of a clock. To this condition the term pendulum rhythm has been applied. It occurs chiefly in nephritis with hypertension. If the heart rate is rapid and the sounds nearly alike the diastolic pause is so shortened that it cannot be distinguished from the short pause between the single sounds. To this condition Stokes[3] gave the name of fetal rhythm. There is no extinction of either sound, according to his view, but both are diminished in loudness, and become of nearly similar character. The name embryocardia was given by Huchard[4] many years later. According to the generally accepted view embryocardia is pendulum rhythm plus tachycardia. H. Müller[5] concludes from a careful clinical study that the so-called embryocardia is due to the extinction of the second heart-sound. This is associated with a fall in blood-pressure. Absence of the second sound explains the similarity of the sounds heard because one hears only the first sound and naturally the interval between the first sound in succeeding cycles is the same if the heart action is regular. Müller maintains that the term embryocardia is bad because the fetal heart never has the pendulum rhythm but the ordinary rhythm of the normal adult heart. He proposes instead cyniclocardia or rabbit heart, because only the first sound of a rabbit's heart is audible. Müller's findings should be confirmed by phonocardiographic study before they can be accepted.

Embryocardia occurs in severe infections, pneumonia, and the terminal stage of tuberculosis. It is of grave omen.

Gallop Rhythm

This is closely related to reduplication of the heart-sounds. It was first described by Bouillard (1835) and he termed it a *bruit de rappel*—a sound of recall or diastolic echo. He was dealing with what seems to have been a reduplication of the second sound. Years later Potain (1875) described an apparent reduplication of the first sound as a *bruit de gallop*.

In gallop rhythm or as the English call it, canter rhythm, three sounds are heard during each cardiac cycle. The intervals between the sounds are approximately equal. The sound closely resembles that made by

[2] Pawinski, *Deutsche med. Wchnschr., Berl. & Leipz.* 1891, XVII, 143.
[3] Stokes, "The Diseases of the Heart and the Aorta," 1854, 379.
[4] Huchard, *Semaine méd., Par.*, 1888, VIII, 186.
[5] Müller, H., *Samml. klin. Vortr., n. F., Innere Med., Leipz.*, 1911, Nr. 197, 198.

the hoofs of a galloping horse. The majority of cases are due to the audibility of the auricular sound (Krehl). Cardiographic tracings in cases of gallop rhythm show either a wave in diastole corresponding to the third sound or an abnormally high auricular wave preceding the systolic elevation. The occurrence of gallop rhythm in the weakened heart of chronic nephritis is ominous. If the heart action is rapid the diastolic period will be so short that it will be impossible to distinguish between the two types of gallop rhythm.

Presystolic Gallop Rhythm.—The phonetic equivalent is te-lub-dub. The accessory sound occurs immediately before the first natural sound. Occasionally it is heard in health (Krehl). It is usually met with in cardiosclerosis, in hypertensive heart disease associated with chronic nephritis, mitral stenosis, hyperthyroidism and acute fevers. An example of this is seen in Fig. 39. It is the more common type of gallop rhythm.

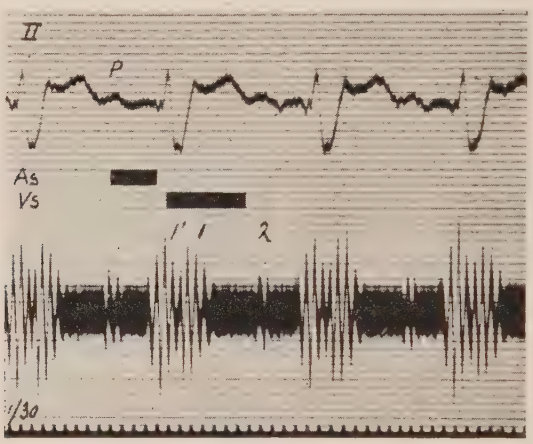


Fig. 39.—Presystolic gallop rhythm at the apex-beat; from a patient who presented evidence of a right bundle branch defect. The first element of the gallop, that is the first of the three sounds lies toward the end of auricular systole. The two elements of the composite sound 1'1 are similar. Both are attributed to the closure of the *A–V* valves; the first being produced by the termination of auricular systole, the second by the onset of ventricular systole. The *As–Vs* interval is a little prolonged. (Lewis, "Lectures on the Heart," Paul B. Hoeber.)

It is evident in this photocardiogram that the first sound is presystolic. Normally there is no interval between the auricular systole and the ventricular systole, but in the case from which the record was made, as in most cases of presystolic gallop rhythm, there is a definite interval between the auricular and ventricular systoles. Normally the auricular sound is blended with the first sound that occurs at the beginning of

ventricular systole, because the ventricular systole immediately follows the systole of the auricle. The pause between the two systoles that occurs when the conduction time from the auricle to the ventricle is increased enables the auricular sound when present to be distinguished from the ventricular sound by a short period of silence. Sometimes the presystolic sound is audible without the interposition of an intersystolic interval. (See Fig. 40.) The explanation is not clear. It probably bears some

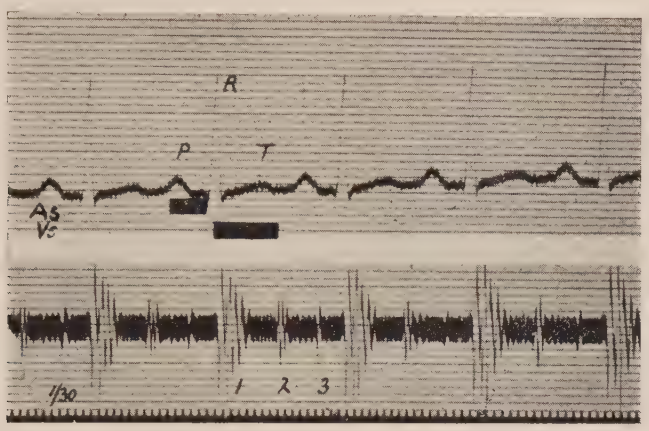


FIG. 40.—Gallop rhythm; the extra sound accompanying auricular contraction. (Lewis, "Lectures on the Heart," Paul B. Hoeber.)

relation to auricular hypertrophy, and may be due to increase in the force of the auricular contraction when ventricular weakness leads to overfilling of the auricle (Moritz, Offenbacher). No case of presystolic gallop rhythm has been seen in which the auricles have been in the state of inactivity associated with fibrillation (Lewis).

Protodiastolic Gallop Rhythm.—The phonetic equivalent is lub-dub-da. The third sound occurs early in diastole. It is usually accompanied by a distinctly palpable diastolic shock (Potain). It is generally regarded as an intensification of the normal third sound, but if the conduction time between auricle and ventricle is greatly delayed it may be caused by the auricular contraction. It is present in some cases of mitral stenosis, aortic insufficiency, and cardiac dilatation with failure in association with chronic nephritis, but it can also occur in a normal heart. In mitral stenosis an early short diastolic murmur may be mistaken for a third sound. If it is a murmur it will be more readily recognized after the patient exercises. In this type of gallop rhythm the third sound is most clearly heard when the heart rate is slow.

The Origin of Murmurs

Any fundamental change in the pitch and quality of the heart-sound or any adventitious sound heard over the heart region constitutes a murmur. A murmur may be defined as a sound produced by the flow of fluid along a tube. The usual condition leading to the production of a murmur is a sudden change in the calibre of a tube. A jet of fluid flowing swiftly out of a narrow orifice into a wider space is called a fluid vein. It was long held that the vibrations of fluid veins were productive of the sounds which, transmitted to the ear, receive the name of murmurs. It is now known that sound does not arise in the fluid, but in the vibratory structures that surround it, namely the walls of the arteries in the case of an arterial murmur; the valves and muscular wall of the heart in the case of a cardiac murmur. It is the transverse vibrations of these structures that gives rise to sound. The longitudinal vibrations produce no sound.

There is no doubt that the moving blood in the arteries and heart disturbs the equilibrium of the surrounding structures, namely, the arterial walls, the muscular walls and valves of the heart, and sets up the vibrations that are audible as murmurs. This is very different from the old view that the sound arose in the blood. The formation of a fluid vein or an eddy gives rise to no vibratory movement. In view of the investigations of Kundt and Lehman the possibility is admitted by Geigel that sound might be produced in the blood within the heart and arteries. But the latter shows by a mathematical calculation that the heart would have to contain 24 liters in order to produce a murmur of the same pitch as the first heart-sound. There is one exception to the general rule that murmurs arise from the transverse vibrations of the walls of the channels and chambers that contain the blood. It is probable that musical murmurs and possibly some very high-pitched whistling or piping murmurs may arise from vibrations of the blood itself. The pitch of these rare murmurs is so high that they could be produced in columns of blood of no greater dimensions than occur in the human body. Some musical heart murmurs are quite likely due to the vibrations of tendinous cords that run an abnormal course across the cavity of the ventricle (C. Gerhardt).

Present knowledge of the origin of murmurs is chiefly derived from the observations of Kiwisch and those of Theodor Weber. It should be pointed out that the Englishman Williams was the first to recognize that

murmurs were due to the vibrations of the walls of the tube containing the moving fluid.

The following statements are based chiefly on the conclusions drawn by Theodor Weber from his experimental studies on tubes through which streams of water were passed. The ease with which murmurs arise and the character of the murmurs depend chiefly on the form of the tube and the nature of its walls and on the velocity of the moving fluid, less on the quality of the fluid. This law of Weber can be made clear by simple explanations. A tube could be of the same diameter throughout or it could have narrowed or dilated portions. The shape of a tube that most readily gives rise to murmurs is one with a narrowing at one point or in medical terminology a tube with a stenosis. The walls might be composed of elastic material, as when rubber tubing or an artery are used, or be rigid, as is of course the case with glass or metal tubing. The fluid used might be water, blood or mercury.

Murmurs occur more readily when the walls of the tube are thin than when they are thick. Murmurs arise the more readily in wide than in narrow tubes. Murmurs arise the more readily the greater the surface area of contact between the fluid and the tube.

The fluid must move with much greater velocity to give rise to a murmur in an unyielding tube like glass than in one that is flexible and distensible, for example, rubber, intestine, artery or vein.

The ease with which murmurs form is dependent in part on the nature of the fluid. They form more readily in mercury than in water, more readily in water than in water mixed with blood, more readily in blood thinned with water than in pure blood.

Murmurs can arise in straight tubes of uniform calibre, but the velocity of the current must be much greater than when a stenosis exists. In the case of a straight, smooth-walled tube the murmur is heard along the entire length of the tube, while a murmur produced by a constriction of the lumen of a tube is heard with greatest intensity over the narrowed place. It should be emphasized, however, that no tube or vessel is absolutely smooth. The slight roughening of the inner walls is equivalent to minute stenoses.

In tubes narrowed at one place murmurs arise most easily, and only a very slight velocity of the stream is necessary for their production. There is a certain degree of narrowing of the tube that is most favorable for murmur formation. The murmur in a tube narrowed at one part is loudest at the point where the fluid emerges from the narrow into the wider portion of the tube. Sometimes the vibrations are so strong that

they can be felt with the finger. They produce a thrill. The sensation is similar to that caused by letting sand fall on the finger. If the velocity of the fluid in the tube is gradually slowed a point is finally reached when the velocity becomes insufficient to produce a murmur.

It was Corrigan [6] the great Irish clinician, who found by experimental study that the condition that most favored the production of murmurs was the passage of fluid from a narrow into a wider space. This received its correct physical explanation from Kiwisch [7] (1850). Unfortunately many years passed before his work became generally known. He showed that the flow of the fluid through the narrowed lumen set up transverse vibrations in the walls of the tube which are audible as a murmur.

The outer layer of fluid as it leaves the narrowed place and enters into the wider portion of the tube does not continue its course in a straight line, but is deflected outward toward the wall of the wider section of the tube. This is due to the adhesion of the fluid to the wall being stronger than the cohesion of the fluid particles. This deviation of the outer layer leads to a similar movement by the adjoining layers of fluid. As a result there is a partial vacuum formed in the centre of the lumen. This has been determined by manometric measurements. As the pressure within the tube is diminished the pressure of the air on the outside of the tube forces the elastic wall inward. The elasticity of the wall offers increasing resistance to this compression. The inward movement of the wall ceases and its elasticity causes it to widen again. Thus the tube becomes alternately compressed by the pressure of the air and expanded by its own elasticity. Thus the horizontal vibrations are set up which are audible as murmurs.

Vibrations are also set up in the narrow portion of the tube above the junction with the wider part. Inward and outward movement of the walls of the wider portion produce synchronously a decreasing and an increasing suction on the stream of fluid. As a result of this, sometimes less fluid and sometimes more fluid flows from the narrowed upper part of the tube. This variation in flow varies the pressure and the consequence must be that the wall of the narrow part is set into transverse vibrations.

The flow of fluid from a wide into a narrow channel can also produce a murmur. The obstruction to the outflow at this narrow orifice raises the pressure in the wider portion of the tube. This increases the pressure

[6] Corrigan, "Inquiry into the Causes of *Bruit de Soufflet* and *Fremissement Cataire*," *Lancet, Lond.*, April 4, 1829.

[7] Kiwisch, "Neue Forschungen über die Schallerzeugung in den Kreislaufsorganen," *Verhandl. d. phys.-med. Gesellsch. zu Würzb.*, 1, 6.

at the stenotic opening with the result that the narrow channel is temporarily widened. More blood flows through it. The pressure in the wider portion falls. Such alternations of pressure throw the vascular walls in both the wide and narrow sections into transverse vibrations.

Instances of murmurs arising from the flow of fluid from a narrower into a wider portion of the vascular bed are common in disease. It is seen in many valvular defects and in aneurism.

Another form of murmur is that due to the vibrations of an elastic diaphragm. This is seen in disease sometimes when a valve segment is partly torn from its attachments and forms a partial obstruction to the onward flow of blood. The increasing pressure produced by the damming back of the blood above the valve segment pushes the segment aside. This gives a wider opening for the passage of blood. The pressure as a result of the freer movement of the blood falls. This causes the valve segment to drop back into its former position, when it again narrows the lumen. As a result of these changes in pressure the valve, flapping like a sail in the wind, may be thrown into audible vibrations.

Intensity of Murmurs

The greater the stenosis up to a certain degree, other conditions being equal, the louder the murmur. This is true both of stenosis and incompetence of the valves. Hence a fairly high degree of narrowing of an orifice or a slight incompetency will give louder murmurs than a slight narrowing of an orifice or a great incompetency. For example, if the aortic valves close except for a narrow slit the high pressure in the aorta will force the blood through the narrow opening and produce a relatively loud murmur. If the aortic valves are so damaged that the opening at the orifice is large, when the valves are closed the blood will regurgitate freely into the ventricle and the murmur will be less loud than when the opening is smaller.

Experimental study shows that narrowing a tube up to a certain degree increases the intensity of a murmur, further narrowing causes the murmur to become less loud. This explains the clinical finding that extreme stenosis of an orifice produces murmurs less loud than stenosis that is not so great. In regurgitation the cleft that permits the backflow of blood has to be of a certain size in order to give rise to a loud murmur, otherwise the less the regurgitation the louder would be the murmur and every-day clinical experience proves that this is not so.

The existence of a narrowing of the stream-bed in itself is not suf-

ficient for the development of a murmur. In addition a certain velocity of the blood-current is necessary. The greater the velocity, the louder the murmur. The velocity is dependent on the power developed during systole by the muscular walls of the different chambers of the heart. If weakness of the heart muscle occurs, the systolic power will be less, the velocity of the blood through the stenosed orifice will decrease and as a result the murmur will weaken or disappear. A good example of the effect of weakening of the muscular power is seen frequently in mitral stenosis. When the auricle is contracting normally a loud murmur is heard. This is due to the contraction of the auricle and it occurs just before the first sound. It is an intense and very characteristic murmur. With the onset of fibrillation of the auricle, the auricle no longer contracts and this murmur disappears.

Excitement and apprehension by quickening the heart's action often cause murmurs to become louder. The fact that the murmur becomes louder when the blood flow is more rapid is utilized in diagnosis. Exercise accelerates greatly the blood flow, and a murmur that is faint or indefinite when the subject is quiet often becomes loud after a few minutes' exercise. The effect of increasing the blood flow by exercise on the development of murmurs should be tested in every routine examination of the heart if the patient's condition permits. The position of the patient affects the intensity of the murmur. Many mitral murmurs are only audible in the recumbent posture. Some aortic murmurs on the other hand are only heard when the subject is standing.

Murmurs vary greatly in intensity. Occasionally they are so loud that they are plainly heard some distance from the patient's chest. Very loud murmurs are rare, and usually occur in aortic insufficiency. They are called distance murmurs. Often these loud murmurs have a musical quality. The murmurs of mitral stenosis are generally weak.

Murmurs as a rule are of longer duration than heart-sounds. The relatively long duration permits the ear to perceive variations in the intensity of the murmur during its course. Usually a murmur is loudest at its beginning and gradually becomes fainter. This is termed a *decrescendo murmur*. A murmur may begin softly and steadily increase in loudness. This is a *crescendo murmur*. Again a murmur may be loud at its onset and gradually become fainter until near the end when it increases in loudness. This variation in intensity is helpful in diagnosis. The murmur of mitral stenosis is regularly of the crescendo type unless auricular fibrillation is present. The murmur begins in late diastole and increases in intensity. It is due to systole of the auricle forcing blood at increased

speed through the narrowed orifice. As soon as auricular fibrillation develops the crescendo character is lost.

The Acoustic Quality of Murmurs

Much stress was formerly laid on the character of murmurs, whether soft, blowing, rasping, rough or purring. According to Gee these are "vain distinctions." The quality is certainly less important than the time and place of a murmur, but may be of some value in diagnosis. More or less unconsciously physicians do lay weight, and justly so, on the character of the murmur, especially in aortic insufficiency and mitral stenosis. In aortic insufficiency in the majority of cases there is a soft long-drawn blowing murmur. In mitral stenosis the presystolic murmur has a harsh vibratory quality and is of shorter duration. It often sounds like a short drum roll. Blowing murmurs may be either low pitched, like the sound produced by a bellows, or high pitched, approaching that of a whistle. Variations in pitch are useful in the identification of murmurs when two or more are present in the same heart. Rough or musical murmurs are usually the result of organic disease. Accidental murmurs, on the other hand, are usually soft and blowing. It is not safe, however, to attempt on quality alone, to distinguish between an organic and a functional murmur.

Time of Murmurs

It is of the first importance in the study of a murmur to ascertain when it occurs in the cardiac cycle. One should note whether the murmur is in systole or diastole. In addition it is important, in case the murmur occupies only a portion of systole or diastole, to determine in what part of the phase it occurs. Systole extends from the beginning of the first heart sound to the beginning of the second sound. Diastole lasts from the beginning of the second sound to the beginning of the first sound of the next cardiac cycle. In mitral insufficiency the murmur occurs early in systole, that is during the isometric period, because, owing to the incomplete closure of the mitral valve, regurgitation begins with the onset of contraction. The murmur of aortic stenosis begins after the isometric period during the expulsion of blood from the ventricle into the aorta. That is .06 to .07 second after the beginning of the first sound. This is a short interval, but long enough possibly to be perceived by a highly

trained ear. It was von Noorden who first recognized the delay in the onset of the systolic murmur in aortic stenosis. In aortic insufficiency the murmur begins with the second sound, but in mitral stenosis the murmur never begins with the second sound, even when it occupies early diastole. The typical murmur of mitral stenosis occurs in late diastole. It is termed presystolic. It is not easy to time murmurs accurately. When instructors claim that they can do so when the heart rate is rapid, students may comfort themselves with the knowledge that their teachers are probably mistaken and are overrating their auditory ability. The discussion regarding the time of the murmur in mitral stenosis supports my statement. Brockbank in 1899 after a careful study concluded that the murmur was systolic and not presystolic. Sahli, a life-long student of auscultation and an eminent clinician and teacher, in the latest edition of his textbook (1920) supports Brockbank's view. The question was only settled by the phonocardiographic study of Lewis which showed that the murmur was presystolic.

A single endocardial murmur is always confined to a single phase of the cardiac cycle. If a single murmur occupies a portion of both systole and diastole it is certainly exocardial in origin (Pericardial friction; patent ductus arteriosus botalli).

Classification of Murmurs

Murmurs according to their place of origin can be divided into two great groups:

1. Murmurs that arise in the heart itself—endocardial murmurs.
2. Murmurs that arise outside of the heart—exocardial murmurs.

The second group can be divided into four classes:

1. Murmurs that develop between the two leaves of the pericardium—pericardial murmurs or friction sounds.
2. Murmurs that develop in the lung—cardiorespiratory murmurs.
3. Murmurs that develop in the arteries.
4. Murmurs that develop in the veins.

Endocardial Murmurs

Disease of the valves of the heart is almost always associated with the development of murmurs. Hence the importance of murmurs in the diagnosis of valvular diseases. Murmurs, however, are frequently present when the valves are healthy.

There are two forms of valvular disease,—the infectious and the de-

generative. The first is common in early life, the second after the age of forty in association with arteriosclerotic changes. One type of infectious endocarditis, and that an important one, forms an exception to the general rule. This is syphilitic endocarditis. It rarely occurs before the thirty-fifth year. These anatomical changes interfere seriously with the function of the affected valve. This occurs in two ways. Either the orifice becomes narrowed (stenosis) or the valve remains partly open when it should normally be tightly closed (regurgitation). In the former case the narrowed orifice interferes with the free flow of the blood onward. In the latter the valve segments fail to close tightly and some blood flows back through the leaky valve.

We have already shown that a narrow place in a tube furnishes the most favorable condition for the formation of a murmur (page 327). Murmurs are produced by a narrowing (stenosis) of an orifice of the heart, and they are also formed when the blood flows backward or, as we say, regurgitates through the opening left by the closure of a damaged valve. It sounds paradoxical to say that murmurs caused by regurgitation through a partly open orifice are due to stenosis. Yet this is true. When the damaged valve is closed there is usually only a small slit open through which the blood passes in its backward flow. Even when valve segments are greatly shrunken the opening will be less wide at the time of closure than it is normally when the valve is open, because it will be partly closed by the damaged valve. It is the regurgitant flow through the narrowed opening that sets up the audible vibrations of the valve and the walls to which it is attached.

I. The Insufficiencies.—Knowledge of the mechanism of the valves and the movement of the blood through the chambers of the heart during the cardiac cycle makes it easy for the student to determine the time in the cycle that the murmurs must occur. He should reason out the time of the murmur in each valvular disease and not simply memorize the relationship. The orifices are open in that phase of the cycle in which they should be closed. The opening is a more or less wide cleft between the segments of the affected valve.

The Auriculoventricular Valves.—(Mitral and tricuspid valves). The valves are normally closed during systole of the ventricle. When mitral or tricuspid insufficiency exists they are partly open at this time. Contraction of the ventricle when both semilunar and cuspid valves should be tightly closed (isometric period) forces the blood in a regurgitant current from the ventricle through the leaky valve into the auricle. This current which causes the murmur occurs during ventricular systole. Hence the murmur must be systolic.

The Semilunar Valves.—(Aortic and pulmonary valves.) These valves are normally closed during diastole of the ventricle. If they are partly open aortic or pulmonary insufficiency exists. Recoil of the arterial walls at a time when the semilunar valves should be closed forces the blood in a regurgitant current from the artery into the ventricle. The current of blood through the narrowed, but not completely closed, orifice causes a murmur. This backward flow occurs at the beginning of diastole. Hence the murmur must be diastolic.

II. The Stenoses.—The orifices are narrowed, that is partly closed in that phase of the cycle in which they should be open.

The Auriculoventricular Valves.—Narrowing of the orifice between auricle and ventricles impedes the onward flow of blood. The auriculoventricular valves are open during ventricular diastole. The auricle contracts towards the end of diastole. The murmur must be diastolic.

The Semilunar Valves.—The murmur is caused by the flow of blood through the narrowed orifice between ventricle and artery (aorta or pulmonary artery). This flow occurs during the ventricular contraction (isotonic period). The murmur must be systolic.

The Time of the Murmur in the Different Valvular Lesions

Systolic murmurs	Aortic stenosis Pulmonary stenosis Mitral insufficiency Tricispid insufficiency
Diastolic murmurs	Aortic insufficiency Pulmonary insufficiency Mitral stenosis Tricuspid stenosis

Localization of Heart Murmurs

As a systolic or diastolic murmur might arise at any one of the four different orifices it is evident that the time of a murmur alone is little aid in determining the place or origin. It should be pointed out that in the great majority of cases the murmurs arise at the two valves of the left heart. This simplifies the problem. It is usually possible to determine at which valve the murmur arises by noting the point of maximum intensity and the direction of transmission to other parts of the chest. The

areas at which murmurs are best heard are in general those of the valve from which they arise.

1. Systolic murmurs arising at the aortic valve are loudest over the second right interspace. Diastolic murmurs may have their maximum intensity here but are usually best heard along the left border of the sternum in the third and fourth interspaces or behind the middle of the sternum at the level of the third rib.

2. Murmurs arising at the pulmonary area are best heard in the second left interspace.

3. Mitral murmurs are loudest at the apex of the heart.

4. Tricuspid murmurs have their place of maximum intensity at the junction of the fifth right rib with the sternum.

Most murmurs are transmitted for some distance from their place of maximum intensity. As one passes from this area the murmur diminishes in intensity. The propagation of the murmurs is as a rule in the direction of the blood-current that produces the murmur. The aortic systolic murmurs are well transmitted into the subclavian and carotid arteries, and are heard feebly if at all at the apex of the heart. The diastolic murmur arising at the aortic valve is usually louder along the left sternal border than in the second right interspace and is sometimes well heard at the mitral area and in the left axilla. It is rarely audible over the arteries of the neck. The murmur of mitral insufficiency is often loud over the pulmonary area as well as at the apex. In mitral stenosis the murmur is generally confined to a small area at the apex and is rarely transmitted to the base.

Localization of Murmurs in Combined Lesions. Puncta maxima and Puncta minima

Often more than one valvular lesion is present and instead of one murmur being heard two or more are audible over the heart.

If only one valve is diseased with the result that a murmur occurs in systole and another in diastole recognition is usually a simple matter by noting the time of the murmurs. Mitral stenosis is as a rule associated with mitral insufficiency. Hence both a presystolic and a systolic murmur are heard at the mitral area.

If two valves are affected and two murmurs in the same phase of the cardiac cycle are present the recognition of the valvular defects may be difficult. For example, if aortic stenosis and mitral insufficiency exist in the same heart two systolic murmurs will be present. The problem is to

distinguish between this condition and a lesion at one valve in which the murmur is heard both at the base and the apex of the heart. There are two aids in locating the place of origin of each murmur when two or more are heard over the heart.

I. The Quality of the Single Murmurs.—As already stated (page 331) murmurs vary greatly in their character. Some are soft and blowing, others are rough or musical. If two murmurs are heard occupying the same phase of the cycle but differing markedly in their quality, it is highly probable that they arise at different valves. If there is only slight difference in the quality of the murmur at different orifices the conclusion is not justified that two murmurs exist, because the pitch or character of a murmur may be altered in transmission from one part of the cardiac area to another.

II. The Points of Maximum and Minimum Intensity.—A murmur has its maximum intensity at its seat of origin and decreases in intensity in passing from this place. If a systolic murmur is heard at both the aortic area and at the apex, note whether it increases or decreases in loudness in passing from one of these areas to the other. Move the bell of the stethoscope 1 to 2 cm. at a time and focus attention on the loudness of the murmur. If a murmur is of equal loudness at both the base and apex there is probably a valvular lesion at both the aortic and mitral valves. The only exception is a murmur of such great intensity that it is heard with equal loudness at both orifices. If the murmur gradually grows fainter in passing from the apex to the aorta then one concludes that the murmur originates at the mitral valve and mitral insufficiency exists. This decrease in intensity is symbolically represented in Fig. 41, I. The width of the figure at any point in its length represents the intensity of the murmur.

The murmur may steadily grow weaker in passing from the mitral area to the aortic area until about midway between the two and then gradually increase in intensity until the aortic is reached when a second maximum is attained. This is represented graphically in Fig. 41, II. This finding can be interpreted as due to the presence of two murmurs, one at the aortic and one at the mitral valve. It is called an "hour-glass" murmur and usually means a double point of origin. Often, however, a murmur that arises at the aortic area is heard more loudly at the aortic area and at the apex than over the body of the heart. This is probably due to the presence of the right ventricle in front of the left ventricle. The left ventricle it will be remembered approaches the surface only at the apex. "A murmur may travel some distance under ground and emerge with a change of quality." (Allbutt.)

If a systolic murmur is heard not only at the mitral and aortic areas but as is usually the case also at the tricuspid and pulmonary areas the question must be settled whether the murmur heard at these areas arises at the corresponding valve (autochthonous murmur) or is simply transmitted. In addition to noting any difference in the quality of the murmurs one should carefully auscultate along the lines AP and AT (Fig. 41, III and IV), extending from the aortic area to the pulmonary and to the tricuspid. Then from the mitral area to the tricuspid area MT and finally from the mitral to the pulmonary MP. If the result is that depicted in Fig. 41, III, in which the puncta minima are at the pulmonary

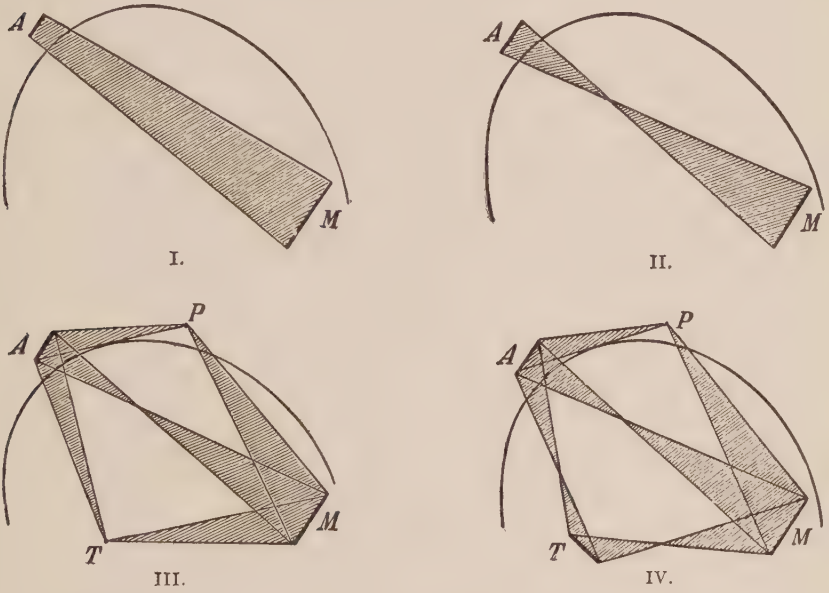


FIG. 41.—Diagram of the relations of transmission of cardiac murmurs. Maximum and minimum points. A. Aortic area. M. Mitral area. P. Pulmonary area. T. Tricuspid area. (Sahli "Diagnostic Methods," W. B. Saunders Company.)

and tricuspid areas it is evident that the murmurs are simply transmitted to these areas. In Fig. 41, IV, the condition is pictured in which there is an autochthonous murmur at the tricuspid area as well as at the mitral and aortic, while the murmur at the pulmonary area is simply conducted there.

It is important for the student to observe carefully the transmission of murmurs and the other characteristics described, but a note of warning is necessary. *No satisfactory diagnosis can be made from murmurs alone. A systolic murmur, no matter how loudly heard at the aorta or mitral*

area and how poorly transmitted to the other valvular areas, does not justify the diagnosis of a diseased valve unless supported by other evidence.

Diastolic murmurs are far more trustworthy guides to the existence of valvular disease than systolic murmurs, but the results obtained by auscultation must always be interpreted in the light of the facts furnished by the other methods of examination, as well as by the case history.

Physiological Murmurs

Systolic murmurs may be present in hearts which are perfectly healthy. Thousands of persons are to-day laboring under the mistaken idea that they have heart disease because some physician has discovered a murmur on which he, in the absence of any other sign of heart disease, has made a diagnosis of valvular disease. Physiological murmurs are most common during the second and third decade. In young people they are usually heard with maximum intensity over the pulmonary area, but may be located at the mitral or aortic areas. If the murmur is loud it is frequently transmitted from the pulmonary to the aortic and mitral areas. As a rule it is only heard in the second left interspace and only over a circumscribed spot. In adults they may have their maximum intensity at the mitral region, but usually in adults as in children the murmur is loudest at the pulmonary area. This has been aptly termed "the region of romance on account of the variety of interpretations given to the murmurs having their position of maximum intensity in that position (Balfour).[8]" Although murmurs in this region are very common, disease of the pulmonary valve is very rare. A systolic murmur due to a lesion of the pulmonary valve is practically never met with except in congenital heart disease. The physiological murmur is usually louder in expiration and is often plainly heard only then. It is more often heard in the recumbent than in the erect posture. Lee found that 70 per cent of college students examined at Harvard had a systolic murmur in the recumbent position after deep expiration. It is transitory generally, but sometimes is constantly present for a period of years. The murmur is increased by exercise and in a large proportion of the cases is only present after physical exertion or during excitement. Forcible and especially rapid heart action increases the loudness of the murmur. The presence of the murmur is influenced by the shape of the chest. It is very common in

[8] "Clinical Lectures on Diseases of the Heart and the Aorta," third edition, London, 1898, 127.

shallow chested persons and rare in those with a large sternovertebral diameter.

The cause of these murmurs is not definitely known. It is probable that many of them, in children at least, are due to a physiological stenosis of the pulmonary orifice (Lüthje). The murmur is most common between the tenth and fourteenth years. At this time the thorax is shallower than at an earlier or later age. The greater loudness of the murmur in expiration is explained by increased pressure on the pulmonary orifice exerted by the surrounding tissues during this phase of respiration. It has long been known that when the chest is thin and elastic, pressure of a stethoscope over the pulmonary orifice might produce a murmur. Physiological murmurs are distinguished from those due to valvular disease by the normal size of the heart, the undiminished reserve power of the heart and the absence of a history of recent rheumatic infection. The separation of physiological murmurs from accidental murmurs is less important. Both are usually classed together. But it seems better to consider as physiological those that occur in the absence of any disease.

Accidental Murmurs

In fevers, in anemias and in other diseases transitory systolic murmurs are frequently present in the absence of any disease of the heart valves or myocardium. They are most frequently located over the pulmonary and mitral areas. These were formerly called anemic murmurs, but the term is an unhappy one as similar murmurs are present in other diseases and in some cases of anemia they are absent. They are especially frequent in chlorosis and in other forms of severe anemia. Hope and Vernois showed as early as 1831 that the withdrawal of large amounts of blood from normal animals caused the development of systolic murmurs over the heart. This experiment does not prove that the murmurs are due directly to anemia. Similar systolic murmurs can be produced in rabbits without bleeding them simply by bodily movements. These exercise murmurs are due to the increased velocity of the blood-current.

The murmurs associated with anemia in man are probably caused in part by the increased rate of blood flow in this condition and in part by the more watery constitution of the blood. A more rapid circulation when the hemoglobin is greatly diminished is a physiological necessity in order to supply the tissues of the anemic patient with a sufficient amount of oxygen.

Diastolic accidental murmurs undoubtedly occur although very rare. In location and character they resemble the murmurs of aortic insufficiency of valvular origin. They have been found only in pernicious anemia. They are thought by Sahli to be simply a venous hum with accentuation of the diastolic part, transmitted from the vessels of the neck and Cabot and Locke who reported three cases share this view. Ortner (1923) believes they may result from a muscular insufficiency at the aortic orifice. The closure of the aortic valve is effected in part by the valve segments, and in part by the contraction of small masses of muscular tissue near the attachment of the valves. In Ortner's cases there was extensive fatty degeneration of the muscular structures, and it may be they failed to function properly as he maintains. If this explanation is correct these murmurs should be classed in the group due to muscular insufficiency.

There is nothing about the quality of accidental murmurs to distinguish them from those due to valvular disease. The student should remember that except in pernicious anemia they are always systolic. If a diastolic murmur is present, any systolic murmur heard is probably not accidental, but due either to organic valvular disease or to muscular insufficiency.

Murmurs of Muscular Insufficiency

These are usually grouped with the accidental murmurs, but as they result from pathological changes in the heart muscle it is better to consider them separately. They are almost always systolic in time and occur most frequently at the mitral valve. They are due to weakness of the muscular ring at the mitral and tricuspid orifices and of the papillary muscles. The imperfect contraction of these groups of muscles permits blood to regurgitate into the auricle even when the valve segments are intact. Murmurs may also be caused by dilatation of the muscular ring to which the valve leaflets are attached. The dilated orifice is then so large that the valve segments cannot close it. This second type of muscular insufficiency is called *relative* insufficiency. Romberg reserves the term muscular insufficiency for the cases of the first group, in which the muscles at the base of the heart and the papillary muscles do not contract sufficiently. Relative insufficiencies are less common than true muscular insufficiencies (Magnus-Alsleben). Muscular insufficiency of the aortic and pulmonary valve is very rare. This is doubtless because the function of the muscular tissue is less important in the closure of these orifices than it is of the mitral.

The murmurs of muscular and relative insufficiency are usually soft and blowing, but sometimes loud and rough. They are often transitory and are especially common in cardiosclerosis. I have frequently seen them disappear when the heart muscle gains in strength. They may be present, however, when the heart reserve is good. They are usually louder in the recumbent position and some may only be heard when the patient is lying down. In persons over forty years of age the systolic murmur of muscular insufficiency of the mitral valve occurs more frequently than any other murmur.

These murmurs of muscular insufficiency and the accidental murmurs are often called *functional murmurs* in distinction from the *organic murmurs* due to valvular disease. But all murmurs are organic in the sense that they have a physical basis; that is, they are all physical phenomena. Cohn [9] has tried to distinguish the two groups by calling one *important* and the other *unimportant*. It is probable that the systolic murmur at the mitral area in acute endocarditis is due largely or entirely to muscular insufficiency. In many cases of acute endocarditis observed at the autopsy table the changes in the valves are too slight to produce an insufficiency yet a distinct murmur had developed during the course of the disease. In such cases the murmur is attributed to weakening of the muscular ring at the mitral orifice or of the papillary muscles as a result of acute inflammation or toxic degeneration of these muscular structures.

Tricuspid regurgitation is usually due to weakness of the muscular supports of the tricuspid valve, very rarely to endocarditis. Mitral insufficiency usually coexists. The tricuspid murmur is heard with maximum intensity over the lower sternum and often differs in quality from the mitral murmur. Signs pointing to regurgitation of blood into the liver and jugular veins are a great aid in the diagnosis of tricuspid insufficiency.

Gibson and others have claimed that muscular insufficiency of the pulmonary valve occurs in certain cases of high pressure in the pulmonary artery and cases have been reported in which this diagnosis was supported by autopsy findings (Graham Steell murmur). In all the cases the right ventricle was dilated. Pawinski attributes the diastolic murmur in the upper half of the thorax in mitral stenosis to pulmonary insufficiency. Goodman claims that he heard it in 12 out of 36 cases of mitral stenosis. Leube on the other hand doubts the occurrence of functional or muscular diastolic murmurs. It is difficult if not impossible to distinguish the murmur of pulmonary insufficiency

[9] *J. Am. M. Ass., Chicago,* 1918, LXXI, 2132.

from that of aortic insufficiency. As the vascular signs may be absent in mild cases of aortic insufficiency I do not believe the diagnosis of pulmonary insufficiency can be made with any degree of positiveness, especially if mitral stenosis exists. In the study of this subject great uncertainty exists.

Exocardial Murmurs

Murmurs in the region of the heart, but produced outside of it, are termed exocardial. Their place of origin may be the pericardium, the substernal tissues, the pleura, the lung, and the arteries of the thorax.

Pericardial Murmurs or Friction-Sounds

These form the most important class of exocardial murmurs. Their origin is similar to that of the sounds produced by the rubbing together of the surfaces of the parietal and visceral pleura when roughened by fibrinous exudate.

The usual cause of a pericardial murmur is a fibrinous exudate on the surface of the pericardium, but it may also be produced by the presence of tubercles or cancerous nodules. It is dependent on the movement of the heart, but not on the closure of the valves.

A pericardial murmur is distinguished from an endocardial murmur by the following characteristics.

(1) It is usually a rough, scratching or creaking sound that is not sharply confined either to systole or diastole.

(2) It seems close to the ear of the examiner.

(3) The murmur is usually divided into two phases, so that it is described as a "to and fro" sound, corresponding to the contraction and dilatation of the heart. Sometimes it is a triple sound like a gallop rhythm, or it may be separated into four parts. When divided into four phases it is called a locomotive murmur. This last form is caused by the movements of the auricles and ventricles when both have fibrinous deposits on their surface.

(4) It is transitory. Its character and its site may vary from day to day or even from hour to hour.

(5) It is not usually located over one of the valve areas. Its most frequent situation is over the upper portion of the sternum and in the third left interspace. The friction-sounds are poorly transmitted as a

rule, and never propagated in definite lines as is the case with endocardial murmurs.

(6) It changes with the position of the patient. It is usually intensified when the patient lies on his left side.

(7) Often the intensity of the murmur can be increased by pressure of the stethoscope, and diminished by inspiration. Some endocardial murmurs are increased by pressure of the stethoscope. (Emerson), but their character is different.

(8) The vibrations are sometimes so intense that they can be felt as well as heard.

In spite of the distinctions that may exist between a pericardial and endocardial murmur in some cases the friction-sound may so closely simulate a murmur of valvular origin that only time can make the diagnosis. The friction-sounds vary in number and extent according to the amount and situation of the fibrinous deposits. If pericarditis sicca is followed by an exudation the pericardial murmur may persist until the layers of the pericardium are completely separated by fluid. It is frequently still heard when a large amount of exudate has accumulated.

For pericarditis over the left border of the heart one listens for the friction-murmur with the patient lying on the left side; for fibrinous exudate of the right border with the patient on the right side. In doubtful cases have the patient lean forward as the pericardial murmur may only be distinctly audible in this posture.

Extrapericardial Murmurs

(1) Pleuropericardial Murmurs.—These arise in the pleura near the pericardium, usually along the left border of the heart where the pleura overlies the pericardium. They are friction-sounds produced by the movements of the heart and also by movements of the lungs. The respiratory part ceases when the breath is held. The murmur is usually lessened in inspiration and increased in expiration. The pleuropericardial murmurs are a sign of pleurisy not of pericarditis.

(2) Substernal Sounds.—In some normal individuals the heart-sounds just above the xiphoid cartilage have a "peculiar harsh scratching, scraping or crunching quality closely resembling a pericardial friction-sound" (Norris). It has been described as the xiphosternal crunch. It is of short duration and heard during both systole and diastole. Blumer thinks it is due to changes in tension in the areolar tissue in the sterno-

pericardial ligament. It may be easily mistaken for a pericardial friction-sound.

(3) Cardiorespiratory Murmurs (also called cardiopulmonary murmurs and systolic vesicular breathing). Many accidental murmurs are attributed to sounds produced in the lungs by the movements of the heart. In the most common form the murmur is a systolic intensification of ordinary vesicular breathing. The normal murmur of inspiration is broken into two, three, or more short murmurs, each of which accompanies a cardiac systole. This was originally described as systolic vesicular breathing (Wintrich 1854, C. Gerhardt 1856). Cardiorespiratory murmurs are loudest and most frequent near the apex and along the left border of the heart (Lewis). These are also commonly heard below the spine of the left scapula, less often to the right of the sternum and over the right back. They are generally louder during deep inspiration. This is an aid in distinguishing them from endocardial murmurs which become fainter during inspiration. They generally disappear in the recumbent position and when the breath is held. They are almost always heard during systole. Often they occur after the first sound from which they are separated by a distinct interval. They are usually short high-pitched whiffs that begin and end suddenly. In cardiac murmurs vibrations predominate which are of about the same rate as many of those present in the respiratory sound (60 to 80 vibrations per second). This similarity in the pitch of the respiratory sound to that of many endocardial murmurs accounts for the difficulty in distinguishing between the two. Cardiorespiratory murmurs are usually associated with rapid heart action. In differentiating between cardiorespiratory and other accidental murmurs the diagnosis of the former should be confined to murmurs of distinctly superficial quality which are loudest over the lung borders and much diminished or absent over the area of cardiac flatness (Hirschfelder). The area over which the murmur is heard extends towards the sternum in inspiration and away from it in expiration.

The cardiorespiratory murmurs are probably due to increased negative pressure in the lung borders near the heart produced by cardiac systole. The heart becomes smaller during systole and the ingress of air in the neighboring bronchioles is increased owing to the greater negative pressure. The sudden increase in the movement of the air in the respiratory passages leads to the production of sound. The subject has been studied most thoroughly by Potain. He showed that the cardiorespiratory murmurs are loudest where the movement of the heart is greatest. He found furthermore that when and where a cardiorespiratory murmur occurred the cardiogram showed a depression. This fall in the curve

indicated a retraction of the chest wall, and was due to an area of negative pressure within the chest. Potain's cardiograms furnished strong support for the theory that cardiorespiratory murmurs are produced by the sudden expansion of air in the lung borders in places where the negative pressure is increased by the systole of the ventricles.

Cardiorespiratory murmurs are so frequently mistaken for the endocardial murmurs of valvular disease that their recognition is a matter of prime importance. During the World War the presence of cardiorespiratory murmurs was the cause of the rejection of many thousand recruits with healthy hearts from the military service.

Rare Auscultatory Signs in the Cardiac Region

Precordial Crepitations.—Through rupture of the alveoli air enters the interstitial tissue of the lung and passes from this through the hilus of the lung into the connective tissue of the anterior mediastinum. The mediastinal emphysema thus produced has a hyperresonant or tympanitic note on percussion, and a crackling sound on auscultation which is synchronous with the heart action.

Pericardial Succussion-Sounds.—The simultaneous presence of air and fluid in the pericardial sac gives rise to a splashing, gurgling, churning sound (sero or pyopneumopericardium). It corresponds to the Hippocratic succussion-sound of sero or pyopneumothorax. Similar, but weaker sounds heard over the heart are sometimes produced in neighboring cavities in the lungs or in a stomach greatly distended with air and fluid.

THE ARTERIES

Inspection

In thin normal subjects a visible pulsation is often present in the carotid arteries and less frequently in the subclavians. It is increased by excitement. Pulsation in other arteries is rarely seen in health. Marked pulsation of the carotids occurs in aortic insufficiency and in exophthalmic goitre. Occasionally the pulsation of the aorta may be visible in the suprasternal notch in the absence of disease. In dilatation of the aorta a pulsation in this location is common, and also when there is a kinking of the aorta in association with a big heart.

Aortic pulsation in the second right interspace occurs:

(1) in certain subjects with normal aortas during periods of excitement;

(2) in aortic insufficiency;

(3) in aneurism of the ascending aorta. It is most marked in this disease and the pulsation is often visible also in the third right interspace.

In aortic insufficiency there is often marked visible pulsation in many of the superficial arteries, including the carotids, brachials, radials and ulnars. Less marked pulsation of the peripheral arteries occurs in hypertrophy of the left ventricle from other causes and in exophthalmic goitre.

In advanced arteriosclerosis in individuals with a scanty amount of subcutaneous fat the peripheral arteries may stand out like cords. The temporals, brachials and radials are especially prominent and are often tortuous. The twisting and turning of the arteries in their course, notably the temporals and brachials, may be of high degree. The increased length of the artery results, according to Geigel,[10] from the action of the flow of blood—not the blood-pressure—on a vessel wall that has lost much of its elasticity. A continuous traction is exerted in the direction of the blood stream and this lengthens the vessel. The ends of the artery being fixed, as its length increases it necessarily becomes curved. Tortuous arteries may move laterally with each heart beat.

Palpation

Palpation of the radial arteries is an important part of the physical examination. In former times the opinion of physicians regarding the state of the heart and circulation was largely based on the information they could gain by feeling the pulse.

The radial artery is felt at the wrist between the styloid process and the tendons of the long flexors of the fingers. The artery is lightly pressed against the radius with the tips of two or three fingers. It is preferable to form the habit of using three fingers (Broadbent). The thumb is not to be used. The wrist of the patient should be supported and relaxed.

In certain cases the brachials, femorals, and other arteries should be examined. The brachial artery is accessible along the entire length of the upper arm. In feeling for the artery in the middle of the upper arm the tips of the fingers are thrust under the biceps from the inner side, so as gently to lift the muscle with the dorsum of the phalanx, whilst the pulp exerts

[10] Geigel, *München. med. Wchnschr.*, 1923, LXX, 564. See also Winkler, *Ibid.*, 1923, LXXI, 1023.

pressure on the artery and on the humerus behind the artery (Ewart). Although the brachial artery is usually palpated in the middle of the upper arm, in many cases it can be more readily felt just above the elbow to the inner side of the biceps tendon. The arm should be flexed at the elbow. The pulsation of the carotid is most easily found at the level of the cricoid cartilage and close to its side. The tip of the finger should be pressed backward towards the spine. The femoral artery is felt just below Poupart's ligament.

When it is important to disturb a patient as little as possible, for example, during sleep, the pulse can be taken in the temporal artery. Find the artery as it crosses the upper edge of the zygomatic process. The further course of the artery is superficial, and easily felt. It is necessary to find this beat quickly in an emergency, or during the administration of an anesthetic and every student should be trained to do so (Ewart). This artery may also be used instead of the radial in cases in which the hand is kept in constant motion such as chorea or mania (Hare).

Condition of the Walls of the Artery.—Arteries of normal thickness and of normal tone cannot be distinguished from the surrounding tissues by palpation. To palpate satisfactorily the vessel wall the radial artery should be emptied of blood. This is done by obliterating the lumen by the pressure of the palpating finger nearest the heart or of one or two fingers of the other hand. The empty vessel is then rolled against the radius with the tips of the palpating fingers. If the empty artery cannot be distinguished from the surrounding tissues it is described as non-palpable. It may be palpable without giving the impression to the finger of being thickened. In cases of chronic nephritis and arteriosclerosis the radial artery often feels like a cord when rolled under the fingers. If the thickening of the wall is of high degree as occurs when the media is uniformly infiltrated with lime salts the artery has been aptly compared to a pipe stem. Small calcareous deposits scattered along the wall of the vessel give rise to the "goose neck" artery, from its similarity on palpation to the trachea of a bird. A calcified artery sometimes feels like a string of pearls.

Healthy arteries in subjects under twenty years of age are rarely if ever palpable. Whether normal radials in older persons are sometimes palpable is a mooted point. Thayer and Fabyan hold that they are. If they refer to anatomical integrity they are doubtless correct in their conclusion that all palpable arteries are not the seat of arteriosclerosis. But if the palpability be due to an abnormal increase in the tonus of the arterial musculature the vessel is then pathological functionally if not anatomically.

Thayer and Fabyan [11] concluded from their anatomical investigation that a palpable thickening of the radial artery was due to sclerotic changes in the vessel especially a thickening of the intima. It is now known, however, that in arteriosclerosis of the radial and other arteries of the extremities the chief lesion is not in the intima, but in the media. It consists according to Aschoff and Marchard in a fatty degeneration of the media followed by calcification. This usually occurs only in the aged and is the cause of the "goose neck" artery (Mönckeberg).

It is generally taught to-day that when the wall of the radial artery thickens as a result of arteriosclerosis the vessel becomes distinctly palpable. Workers in Romberg's clinic have shown that this is not true. Fischer and Schlayer [12] found that many arteries (radials, carotids, brachials and femorals) that were not at all rigid during life showed on histological examination marked thickening and sclerosis of the intima. On the other hand seventy-five per cent of the arteries which Romberg, a skilled clinician, regarded as moderately or markedly thickened during life were found after death to have no thickening of the intima.

Lande [13] later took up the same problem using an improved technique which prevented the arteries from contracting during the fixing and hardening processes. She likewise found no close relation between the condition of the arterial wall as made out by palpation and the anatomical condition. Not only were vessels with undoubted sclerotic changes not detected during life by Romberg, but in about 50 per cent of the arteries that were palpable the histological examination failed to explain the cause of the finding on palpation.

Romberg [14] in his textbook states that one cannot distinguish between the early stages of thickening due to arteriosclerosis and palpability due to increased active tension of the wall. The latter is due to increased tonus of the arterial musculature and is under the control of the vasomotor system.

If the radials are found to be thickened the examination should be extended to the brachials, carotids and femorals. It should, however, be impressed upon the student that the existence of arteriosclerosis of the radial and other peripheral arteries does not indicate the presence of a general arteriosclerosis. Often the aorta, coronary and cerebral vessels are normal when the radials are sclerosed.

[11] Thayer and Fabyan, *Am. J. M. Sc., Phila.*, 1907, CXXXIV, 811.
[12] Fischer and Schlayer, *Deutsches Arch f. klin. Med., Leipz.*, 1910, XCVIII, 164.
[13] Lande, *Deutsches Arch. f. klin. Med., Leipz.*, 1914, CXVI, 295.
[14] Romberg, "Lehrbuch der Herzens und der Blutgefässe," dritte Aüflage, Stuttgart, 1921, 43.

Comparison of the Pulse in the Two Radial Arteries.—In the normal subject the two radial pulses are of the same size and synchronous. A difference in size is not uncommon and results from a variety of causes. A want of synchronism is rare and of serious import, being usually the result of aneurism. The patient should be seated with the ulnar side of the forearms supported on a table. The observer should place himself opposite the patient and the palpation of the two radials carefully made if it is to be of any value. Check the result obtained by crossing the hands so that the right hand feels the right pulse and the left hand the left pulse. If the same difference is again detected it is probably a real one (Ewald). If a difference in the size of the pulse is discovered determine whether it is of central or peripheral origin by comparing the pulsations in the brachial and subclavian arteries.

Difference in the size of the pulse in the two radials may be due to—(1) anomalies of the artery, (2) aneurism, (3) arteriosclerosis, (4) pressure on an artery from external disease such as a tumor, (5) embolism, (6) thrombosis. In an arm with weaker muscles than the other arm the pulse is often smaller and the artery narrower and of lower tension. The difference is often well marked in a case of old hemiplegia in which the pulse of the paralyzed arm is smaller, the artery narrower and less tense than in the other arm with a better musculature (Romberg). Little significance need be attached to slight differences in the pulse on the two sides of the body.

In comparing the two radial pulses simultaneously it is important to note whether they are synchronous or not. A delay of the beat in one radial is a diagnostic sign of aortic or subclavian aneurism. Associated with the delay there will usually be a smaller artery and a more compressible pulse on the affected side and the vessel will be more continuously full between the beats (Broadbent). The modification of the pulse is well shown in the sphygmogram of the delayed pulse. The upstroke is more sloping, the height less, the summit more rounded and the fall more gradual. The delay of the palpable beat as Marey pointed out is not due to a delay in the onset of the wave, but of the period of maximum pressure, giving rise at times to well-marked anacrotism.

Taking the Pulse.—If the pulse is regular and of normal rate it is sufficient to count the beats in fifteen seconds and multiply the result by four, as the minute is the clinical unit of time. If irregular, or slower than normal, or accelerated, the rate for at least thirty seconds should be counted. Furthermore if the pulse is irregular it is important to count the rate over the apex as well as at the wrist. The difference between the two rates is termed the pulse deficit (James and Hart). In cases of

auricular fibrillation with cardiac weakness a pulse deficit is often present.

The pulse-rate should be taken if possible when the patient is not excited or apprehensive. Celsus advised physicians not to be in a hurry to feel the pulse. It is a good general rule to delay the taking of the pulse until after the examination of the lungs and heart as the initial excitement of the patient is usually then allayed. Opinions differ, however, and one of the wisest physicians of his time, Dr. James Jackson, states in his "Letters to a Young Physician" that he found it "better to make this examination early in the visit, before the patient has become fatigued or excited; and if there seems to be any emotion at that time, the examination may be repeated when the patient has become calm."

The Qualities of the Pulse Wave

The following five qualities of the pulse are to be distinguished:—

(1) Rate or frequency (Pulsus frequens or rarus).
(2) Size or volume (Pulsus magnus or parvus).
(3) Tension or hardness (Pulsus durus or mollis).
(4) Swiftness and slowness of the pulse; or short and long duration of the pulse wave (Pulsus celer or tardus).
(5) Rhythm (Pulsus regularis or irregularis).

Of these qualities, rate, tension and rhythm deserve the most study because of their importance in diagnosis.

Frequency

Variations in the Rate of the Normal Heart. The normal pulse-rate averages 70 in a man and 80 in a woman. At birth it varies from 130 to 140. The rate decreases rapidly in early childhood and then more slowly until sometime between the sixteenth and twentieth year when the rate drops to that of the adult. At ten years of age the normal rate is 90 to 100. In old age the rate may rise somewhat (70 to 90). This acceleration above the age of 60 is by some ascribed to diminishing vagus tone, by others to cardiovascular changes.

Any rate between 60 and 90 pulsations per minute in the adult may be regarded as normal, any rate below 60 as slow, any rate above 90 as accelerated (Ewald).

The pulse is slower in large men than in small men. In some families a slow rate is an inherited characteristic. Some individuals normally have a persistently slow pulse. The most famous instance is that of Napoleon whose pulse rate is stated to have been habitually 40.

There is a diurnal variation in the rate. It is usually highest in the late forenoon. The rate is low when quietly recumbent. Movement increases the rate. It is more rapid when standing than sitting and rises quickly on exercising. The usual rate after a walk at an ordinary pace is 100, after a brisk walk 140, after running rapidly 150 (Nicolai). The pulse-rate varies with the metabolic rate. The pulse is slow before rising in the morning and this corresponds to a low metabolic rate. Dietary restrictions cause a striking reduction of the pulse-rate. In several subjects studied by Benedict and his associates the pulse-rate in the morning before rising was less than 40 per minute. The pulse-rate falls in sleep. Klewitz [15] found an average drop in the rate of 20 beats. Deep inspiration tends to accelerate the pulse and expiration to slow it. Psychic excitement increases the rate. This is demonstrated daily in the examination of new patients, see page 350.

These variations in the rate of the normal heart are due to changes in the rate of impulse formation in the sinus node. Impulse formation is influenced by the vagus and accelerator nerves acting alone or in combination.

Tachycardia with a Physiological Rhythm

Rapid heart action occurs normally during exercise as already stated. The acceleration is more marked in those unaccustomed to physical exertion, and in convalescence from some infectious diseases, for example influenza.

In *fever* the pulse-rate is quickened, usually the rise is 5 or 6 beats for every degree of temperature (Fahrenheit). In certain infections the relation of pulse to temperature is quite different. In typhoid fever the pulse is low in proportion to the fever, especially in the early stage of the disease; while in diphtheria and scarlet fever and tuberculosis, the pulse-rate is relatively high. In pulmonary tuberculosis the pulse-rate is usually rapid even when the temperature is normal.

In nervous states a transitory tachycardia is very common. The pulse-rate in cases of cardiac neurosis or "effort syndrome" taken when patients are up and about usually ranges from 90–100 (Lewis). Exceptionally rates of 120–130 are present for a short time. Slight psychic stimulation or bodily exertion raises the pulse-rate unduly in cardiac neurosis, and the elevated rate persists longer than in health. The rise of the pulse in fevers is due partly to the increased metabolic rate, partly

[15] Klewitz, *Deutsches Arch. f. klin. Med., Leipz.*, 1913, CXII, 38.

to the direct effect of the elevated temperature on the impulse formation in the sinus node, and partly to the specific toxins. The latter affects the pulse-rate quite differently due to variations in the character of the specific poisons in the different infections.

I have seen one case of cardiac neurosis with no evidence of hyperthyroidism in which the pulse was always found between 110 and 120 for a period of two years. The patient was not at rest. Cabot refers to the case of a classmate whose pulse-rate when counted was never less than 120, yet he presented no evidence of disease.

The duration of systole shortens as the pulse-rate increases. It is a little above 0.25 second when the heart-rate is over 100 and between 0.28 and .30 second when the rate is between 66 and 80. In cardiac neuroses the systolic phase is said to be longer than in normal men with equal heart-rates (Wiggers and Clough).[16]

In exophthalmic goitre and in hyperthroidism without exophthalmos the pulse is persistently high. This is partly due to the increased metabolism, but chiefly to the action of the thyroid secretion which probably excites the accelerans cordis.

A rapid pulse-rate is an important symptom of cardiac weakness, both in muscular and valvular disease of the heart. It also occurs in acute endocarditis and pericarditis and in the diagnosis of the former condition a persistently rapid pulse-rate is held by many to be of great importance. Too much significance, however, should not be attached tachycardia in cases of suspected acute endocarditis as any acute toxemia or fever, or nerousness may give a higher pulse-rate than acute endocarditis.[16] The acceleration of the pulse is much more marked in the heart failure associated with aortic insufficiency and mitral diseases than with aortic stenosis. The pulse is also relatively slow in the cardiac weakness of hypertonia. In both aortic stenosis and hypertonia there is increased resistance to the systolic discharge of the contents of the left ventricle. This increased resistance is probably the primary cause of the slower pulse-rate in these conditions although the exact connection between the increase of resistance to the emptying of the ventricle and a slow pulse is not understood. (Edens.)

The pulse is usually very rapid in circulatory collapse and surgical shock. It is small, thready and difficult to count.

Atropine injected subcutaneously causes a rise in the pulse-rate the extent of which is dependent upon the vagus tone. Normally the rate of impulse formation in the sinus node is slowed by a continuous vagus

[16] Wiggers and Clough, *J. Lab. & Clin. M., St. Louis,* 1919, IV, 624.

stimulation. When this is removed by cutting this nerve in animals or by the action of atropine the heart-rate becomes more rapid. The subcutaneous injection of 1/60 grain of atropine in young persons usually raises the pulse-rate to about 130 a minute. Less effect is obtained in old subjects because in them the vagus inhibition of the impulse formation is less than in the young.

In paralysis of the vagus as a result of disease the pulse-rate is rapid. This occurs in the final stage of basilar meningitis. Pressure paralysis of the vagus in its course in the neck or thorax probably can occur. This is the explanation for the very rapid pulse in certain cases of mediastinal tumor (Teschenmacher) and of aneurism (Quincke).

Bradycardia

Slowing of the pulse is due (1) to slowing of the rate of impulse formation in the sinus node (sinus bradycardia); (2) to blocking of the impulse to contraction in its passage from auricle to ventricle. This latter is known as heart-block and will be considered under the disturbances of rhythm.

Sinus bradycardia is brought about by a number of causes and occurs in different diseases. It is a very common occurrence in normal persons.

Digitalis in toxic doses slows the heart-rate by increasing the vagus inhibition of impulse formation in the sinus node. Contrary to the general teaching slowing of the heart-rate rarely occurs when the heart action is regular and the drug given in therapeutic doses. Edens maintains that it is only seen clinically in hearts in which the peripheral endings of the vagus have been "sensitized" in some way by morbid changes in the myocardium (hypertrophy with cardiac weakness). The reputation digitalis has gained for slowing the pulse is due to its remarkable power to slow the rate in auricular fibrillation.

Acute cerebral pressure produces slowing of the heart by its direct action on the vagus center in the medulla. This occurs in the earlier stages of meningitis, and sometimes in fracture of the skull, and cerebral hemorrhagia. (Fig. 42.)

Pressure on the vagus nerve slows the pulse in some individuals even when light pressure is exerted in the neck. Tumors pressing on the vagus may produce a persistent bradycardia. Edens observed a pulse of 38 due to pressure on the vagus by cancerous lymph-nodes near the angle of the jaw.

Ocular pressure. Aschner [17] first observed that pressure on the eyeball caused slowing of the pulse and decrease of respiration. It was also demonstrated that the afferent impulse of the reflex passes through the trigeminal nerve to its nucleus in the midbrain and that the efferent impulse passes by way of the vagus nerve. Levine compared the slowing produced by ocular and direct vagal stimulation and found the former to be more effective.

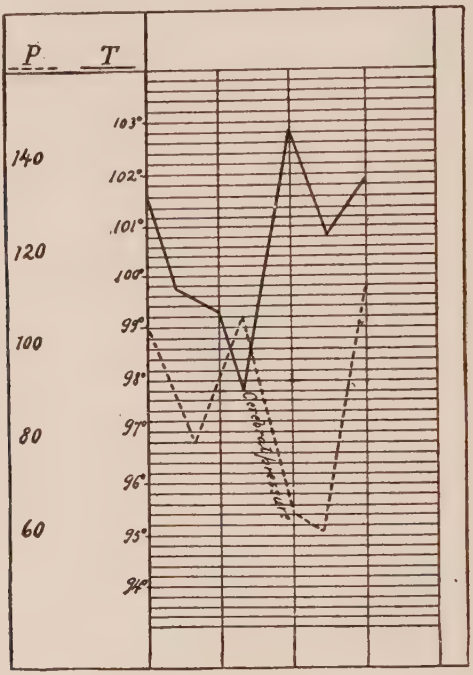


FIG. 42.—The temperature and pulse rate in tuberculous meningitis. The solid line is the temperature record. The interrupted line is the pulse record. (Sahli, "Diagnostic Methods," W. B. Saunders Company.)

Strong emotion may lead to slowing of the heart-rate. Bradycardia accompanies the fainting attacks which occur in neurotic young individuals from emotional causes. The slowing involves both the auricles and ventricles (Lewis and Cotton).

Disease of the vagus center in the medulla. Arteriosclerosis of the vessels of the medulla has been the apparent cause of a persistent slow pulse (Hoffman).[18]

Reduction of metabolism leads to a reduction of the pulse-rate. This

[17] Aschner, *Wien. klin. Wchnschr.*, 1908, XXI, 1529.

[18] Hoffman, *Deutsches Arch. f. klin. Med., Leipz.*, 1910, C. 175, Lit.

occurs in myxedema. The diminution in the thyroid secretion in this disease has an even more marked affect in slowing the heart by its direct action on the heart than does the lowered metabolic rate. Lessening of the thyroid secretion has thus a twofold action in that it slows the metabolism and also the heart-rate. Slowing of the metabolism lowers the heart-rate and lowering the heart-rate reduces still more the metabolism. In this way a vicious circle is established (Edens).

The slow pulse in convalescence from acute febrile diseases is possibly but not probably due to the reduction of the metabolic rate. Another theory is that it results from increased vagus tone or fatigue of accelerator tone.

Jaundice. In certain cases of jaundice the pulse is slowed. This occurs in acute jaundice, particularly in catarrhal jaundice. It is rarely if ever seen in icterus of long duration. It is the result of increased vagal inhibition of stimulus formation. Although usually attributed to the bile salts it is held by King and Stewart [19] to be due to the action of the bile pigments.

Increased resistance to the systolic output of blood lowers the pulse. This is seen in aortic stenosis and sometimes in various forms of hypertonia according to Edens. The slowing of the pulse due to this cause is never marked and the rate becomes more rapid when the heart weakens.

Bradycardia due to vagus stimulation can be distinguished from that due (1) to diminished impulse formation from other causes and (2) to heart-block by the subcutaneous injection of 0.001 gram of atropine. The pulse-rate will become more rapid if the slow heart-rate is due to vagus inhibition as the atropin removes the vagus action by paralyzing the nerve endings in the heart.

Pseudobradycardia. The pulse-rate may be slow in the radial artery in some cases of extrasystole, especially in pulsus bigeminus. This is owing to the failure of weak ventricular contractions (extrasystoles) to send a pulse wave to the wrist. The error of mistaking this condition for true bradycardia is obviated by determining always the rate over the heart as well as in the radial artery.

The Size of the Pulse

The tendons near the radial pulse must be well relaxed if the examiner is to get an adequate idea of the size of the pulse. The arterial pulse is essentially a pressure-pulse rather than a volume-pulse. That is, the

[19] King and Stewart. *J. Exper. M., N. Y.*, 1909, XI, 673.

finger perceives chiefly the difference in pressure between the systolic filling and the diastolic emptying of the artery, rather than the change in volume. The latter is too small to be measured by the palpating finger.

The size of the pulse is dependent on (1) the volume of the systolic output of the heart, (2) the readiness with which the arteries empty themselves during diastole, and (3) the tone of the muscles of the arterial wall. (*a*) The greater the systolic output, (*b*) the more fully the arteries discharge their contents in diastole, (*c*) the more yielding the arterial wall, (*d*) the slower the rate, the greater is the size of the pulse. A tall pulse wave as felt by the finger or seen in the sphygmogram is usually a large pulse.

The pulse is large in aortic insufficiency, in acute hemorrhage and in some fevers. The pulse is small in cardiac weakness, attacks of fainting and during the initial chill of fever.

Tension or Hardness of the Pulse

The artery in different diseases varies in its compressibility. The hardness of the pulse is estimated by the amount of resistance offered to the fingers in obliterating the pulse wave. It corresponds to the mean blood-pressure. A hard pulse is obliterated with difficulty. Normally the pulse is only felt in the radial artery when the pressure is at its maximum during systole; that is during that period of the pulse wave when the pressure is between 100 and 120 mm. of mercury. With a hard pulse the artery is felt during diastole as well as systole because the pulse during diastole does not fall below 100 mm. In fact the minimum pressure in a hard pulse is frequently 120 or more. When the diastolic pressure is high the radial artery feels like a firm cord even when its walls are not thickened. The blood-pressure can be roughly estimated by noting carefully the amount of pressure required to obliterate the pulse in the radial artery with the fingers of one hand above the wrist while the pulse is palpated at the wrist with the fingers of the other hand. Pressure should be exerted by the finger farthest from the heart to prevent reflux of the blood through collaterals. This method does not give accurate results and should be always supplemented by a determination of the blood-pressure with the sphygmomanometer. The pulse is hard in some diseases of the kidneys particularly chronic interstitial nephritis, in primary hypertonia and in lead colic. It is soft in fever, tuberculosis, and usually in cardiac weakness.

Swiftness and Slowness of the Pulse (Pulsus celer and Pulsus tardus)

The designations swift and slow refer to the rapidity with which the palpating finger is elevated by the pulse wave. This elevation is due to the rise of pressure during systole. It is the rate with which the pressure increases to the systolic elevation.

The swift pulse (pulsus celer) is found when a great increase of pressure occurs suddenly. It is present in fever and exophthalmic goitre, but is found in its most pronounced form in aortic insufficiency. When the celerity of the pulse in this disease is present other conditions aid in the suddenness with which the palpating finger is lifted. These are the low blood-pressure in the artery in the intervals between the pulsations, which conveys the impression to the finger that the vessel is empty, and the large size of the artery due to the relaxation of its wall. The sudden collapse of the pulse is often more striking than the rapid rise. Hence it is often called the collapsing or water-hammer pulse or the Corrigan pulse after the great Irish physician, who is credited with having first described it. The wave comes with a "violent rush, filling the vessel and lifting the fingers forcibly. It is as short as it is sudden, and the artery at once collapses again under the pressure of the fingers" (Broadbent). The hand and wrist must be elevated above the elbow and shoulder if these features are to be fully brought out. In cases of slight aortic insufficiency and in cases combined with aortic stenosis celerity of the pulse is not present. The difference between the maximum and minimum blood-pressure (pulse pressure) in the pulsus celer of aortic insufficiency may amount to 80 or 100 mm. of mercury while normally it is only 50 mm.

The slow pulse (pulsus tardus) is due to a slight rise in pressure occurring slowly. It occurs in arteriosclerosis with hypertonia because the diastolic blood-pressure is high and the peripheral resistance, due to the unyielding and contracting arterial walls, prevents a rapid rise of the pressure. It is found in most typical form in aortic stenosis because owing to the narrowing of the aortic orifice the filling of the aorta can only take place slowly.

The swift and slow pulses give characteristic sphygmograms. (See curve e Fig. 49, page 374; Fig. 114, page 454 and Fig. 116, page 461.

Rhythm of the Pulse

The palpating finger detects usually no variation in the interval between the pulse-beats in examining healthy adults. The pulse-rate seems per-

fectly regular. As a matter of fact careful measurements of the pulse waves have shown that a slight difference in the length of the single beats exists in the majority of healthy subjects. A difference was found in 97 per cent of the persons examined as reported by Rehfisch. It is so slight that it may not be recognized on feeling the pulse. The term regular pulse as used clinically refers to the rhythm as judged by the finger.

Auscultation of the Arteries

Normal.—Sounds are heard with the stethoscope over the carotid arteries and frequently over the subclavian, rarely over the femoral. The other arteries are silent in health. In examining the carotid the stethoscope bell may be placed at the angle of the jaw, the level of the larynx or above the inner end of clavicle over the insertion of the sternocleidomastoid muscle. The subclavian is auscultated in the outer portion of the infraclavicular region near the humerus that is known as Mohrenheim's fossa. Two sounds are heard over these arteries, when the stethoscope is lightly applied. The first occurs with the cardiac systole and the second with the cardiac diastole. The first sound arises in the artery and is due to the arterial wall being set in vibration by the rise of pressure and onward movement of blood produced by the ventricular systole. The second sound heard is the second sound of the heart transmitted from its place of origin, the semilunar valves, and occurring immediately after their closure. Usually no sound is audible over the femoral in health, but occasionally one sound, and that the arterial first sound, is present.

If pressure is exerted over these arteries with the stethoscope the normal sounds are replaced by a murmur which is synchronous with the arterial pulse. By increasing the pressure the murmur changes to a clear sound. The pressure murmur is due to the artificial transitory narrowing of the vessel (stenosis) produced by the pressure of the stethoscope. The pressure sound that develops with increase of pressure is caused by the vibrations of the arterial wall in consequence of the high degree of the stenosis.

A systolic murmur may frequently be heard over the anterior fontanelle or in the temporal region of little children between the third month and the fifth year. It probably arises in the internal carotid. Although occurring in healthy infants it is heard with greatest frequency in rickets. It was first described by Fisher of Boston in 1833.

Pathological.—A systolic murmur is often present in aneurism of the aorta but is of little value in diagnosis. The second arterial sound is

often ringing and accentuated in aneurisms of the aortic arch and is an important sign.

In aortic stenosis the systolic murmur that arises at the aortic orifice is well transmitted to the carotid artery. The diastolic murmur of aortic insufficiency is rarely heard well over the carotid and subclavian and the second arterial sound in this disease is usually weak or absent. In aortic insufficiency a sound, cardiac systolic in time, is frequently audible over the brachials, radials and ulnars, arteries which yield no sound in health, and occasionally over the smaller arteries, such as the dorsalis pedis and the arcus volaris. This is caused by the vibration of the arterial wall produced by its abrupt distention during the cardiac systole. This sound is usually so loud and sharp over the femoral artery that it is called the *pistol shot sound*. Sometimes a sound is heard over the femoral in high fever, anemia and chlorosis, the genesis of which is not clear (Luetje).

A *double sound* (Traube's double sound) is occasionally heard over the femoral artery in severe aortic insufficiency. If stronger pressure is made with the stethoscope over this artery a double murmur develops (cardiac systolic and cardiac diastolic). This is known as Duroziez's sign. These two signs—the double sound and the double murmur—probably owe their origin to the sudden and strong stretching of the vessel wall during cardiac systole and the diastolic collapse. The double murmur is heard when the stethoscope produces a stenosis by its pressure. The first murmur is caused by the onward movement of the blood through the narrowed lumen, and the second murmur through the reflux of blood through the stenosed portion during the cardiac diastole. Traube's double sound has been noted also in mitral stenosis, pregnancy and lead poisoning. (Morawitz.)

Loud blowing murmurs are heard over vascular goitres and the pregnant uterus. A systolic murmur is sometimes heard over the subclavian artery in cases of retraction and shrinkage of the pulmonary apices and in diseases of the pleura, but little if any diagnostic significance is attached to it.

Capillary Pulse

Capillaries are in a high degree dilatable. They hold much blood when dilated, and almost exclude blood when contracted. Dilatation of the capillaries produces *flush,* and contraction, *pallor.* Normally capillaries do not pulsate. If they did one would expect that blushing would be intensified with each systole of the heart. (Ewart.)

In some disturbances of the circulation the capillaries may pulsate. In extreme grades of aortic insufficiency the face or hand may blush visibly with each heartbeat. (Osler.) It is obvious that when capillary pulsation occurs the peripheral resistance must have lessened or the force of the systole increased beyond the normal. Dilatation of the arterioles is the main cause of the capillary pulse. It is not easily seen by the untrained, and has to be pointed out to most beginners. It long remained undiscovered probably because the subjects of capillary pulsation are often pallid. The existence of a capillary flush is essential for the productions of the capillary pulse. The observer looks for a waxing and waning of this flush. The pressure of a tightly fitting hat observed by Quincke led him to discover the capillary pulse. A line firmly drawn with the finger across the forehead produces at its margin a zone of hyperemia. On close inspection an alternation of pallor and of injection can be seen if the capillary pulse exists. It is often well demonstrated by the lip method. This consists in pressing gently a glass slide on the everted lower lip. A patch of pallor will be produced. If the pulse is present the margin of this patch alternately blushes and pales. The nail method is also recommended. Gentle pressure is applied to the tip of a finger nail so as to bend it slightly. The hyperemic margin of the pale patch is to be watched for the capillary pulsation. In making these examinations a good light is essential and the observer must shift his position until he finds the one at which the color changes are best perceived.

The capillary pulse is most marked in aortic insufficiency, but also occurs in chlorosis and other severe anemias, occasionally in exophthalmic goitre and in vasomotor neurosis. It has even been met with in healthy persons when the heart action was excited, and sometimes occurs locally in an area of acute inflammation.

THE VEINS

Inspection

Normally the superficial veins of the body are as a rule not distended with blood and are usually invisible. The jugular vein forms an exception. In health it is often seen as a pulsating distended column when a subject is examined in the recumbent position. It is not visible when a healthy person stands erect except occasionally in its lower part just above the clavicle. In inspiration the vein collapses as its contents are emptied into the thorax; in expiration it becomes distended with blood. "Overdistention of the veins is one of the most important physical signs which

we possess of heart disease." (Lewis.) It indicates dilatation of the right auricle.

Jugular Venous Pulse. The rhythmic fluctuations in the size of the vein which follow the movements of the heart constitute the venous pulse. It should not be confused with the carotid pulse. In general the jugular pulse is more readily seen than felt while the carotid is more readily felt than seen. Only in cases of tricuspid regurgitation does the venous pressure rise sufficiently for the vein to feel hard beneath the palpating finger. There are two forms of venous pulse;—the auricular or negative and the ventricular or positive. They are described in the section on the phlebograph.

The Liver Pulse. When the liver is enlarged from chronic passive congestion it often pulsates. On bimanuel palpation, with one hand below the right costal margin and the other pressing forward from behind, the upper hand rises and falls with the pulse. It is a venous pulse and occurs in both the auricular and ventricular form. It most frequently develops in cases of auricular fibrillation. The auricular liver pulse has been observed in a number of cases of tricuspid stenosis, and its presence is strongly suggestive of the existence of this valvular lesion. In great cardiac enlargement the liver may move up and down. This is not a true pulsation of the liver and can readily be distinguished from a liver pulse by means of a polygraphic tracing.

The Progressive or Penetrating Venous Pulse (Quincke)

In rare cases of aortic insufficiency, but occasionally in fever, and even in healthy subjects in hot weather (Vierordt) the arterial pulsation is transmitted through the capillaries into the veins. One then sees pulsations in the small veins on the dorsal surface of the hands and feet. If pressure is exerted on the veins, the pulsation ceases above, that is on the cardiac side of the place compressed, but continues in the peripheral portion. This interesting phenomenon is due to a large and rapidly moving pulse wave (pulsus celer et magnus). A capillary pulse is not always seen in association with it.

The Diastolic Venous Collapse

This consists of the disappearance during diastole of the previously distended veins of the neck. It is an important sign in certain forms of adherent pericardium but is also seen in other conditions. It was first

observed and described by Friedreich. During systole the adhesive bands between the pericardium and the chest wall so act as to prevent the flow of blood from the jugulars into the thoracic veins. In diastole the portion of the chest wall that has been retracted during systole suddenly springs forward, increasing quickly the negative pressure in the thorax and as a result the blood is sucked from the neck veins into the chest.

Paradoxical Movement in the Veins

The veins of the neck emptying into the thorax normally, when visible at all, can be seen to collapse during inspiration and to fill during expiration. Sudden increase of the thoracic pressure, as on coughing, causes the veins to swell. This is especially well seen in the external jugulars. A paradoxical behavior may occur in adherent pericardium. Adhesions or bands by narrowing the innominate vein or the superior vena cava in inspiration dam back the blood in the jugulars. As a result they swell during inspiration and empty during expiration. Its causation is then similar to that of the mechanical form of pulsus paradoxus, and the two physical signs are usually found in the same case.

Auscultation of the Veins

A continuous murmur is often heard over the jugular veins, especially the right, in chlorosis and other severe anemias. It resembles a humming top and is called the venous hum or the *bruit de diable*. Light pressure with the stethoscope sometimes brings out the murmur in healthy persons but it is heard in chlorotic patients when no pressure is exerted (Gee). The murmur in health is only a soft blowing sound while in chlorosis it is a loud murmur that may be so intense that it is palpable as well as audible. To obtain the venous hum Flint directed that the patient should turn his head far to the left and the stethoscope be applied with varying degrees of force to the right side of the neck outside the sternocleidomastoid muscle and near the clavicle. Care should be taken not to mistake a stenotic murmur produced by pressure for a venous hum. More important in diagnosis than the murmur in the jugular is the venous hum transmitted from the intrathoracic veins to the upper part of the anterior chest. This is often heard in the first and second interspaces to the right of the sternum in severe anemia. The diastolic portion of this murmur is intensified and when heard at the aortic area is easily mistaken for the murmur of aortic insufficiency. Sahli believes that the diastolic

murmur in cases of so-called functional aortic insufficiency without valvular lesion, occurring in pernicious anemia, is nothing more than a venous hum. During inspiration and in the upright position the venous hum becomes louder. The murmur is probably produced on the right side of the neck by the rapid flow of blood from the relatively narrow jugular vein into the bulb of the jugular. It is then a form of stenotic murmur, and it occurs in anemia because the velocity of the thinned blood is greatly increased in this disease.

CHAPTER III

INSTRUMENTAL METHODS EMPLOYED AS AIDS TO THE PHYSICAL DIAGNOSIS OF DISEASES OF THE HEART

BLOOD-PRESSURE DETERMINATIONS OR SPHYGMOMANOMETRY

The estimation of the blood-pressure in routine clinical examinations dates from the invention by Riva-Rocci about twenty-five years ago of a simple apparatus. The mercurial instruments in general use to-day are modifications of the one designed by Riva-Rocci. See Fig. 43. The

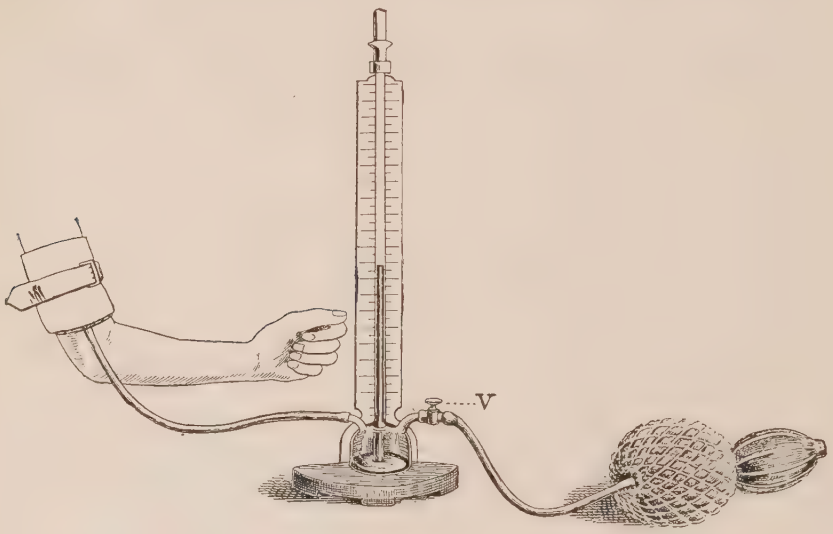


FIG. 43.—Riva-Rocci sphygmomanometer. (Krause, "Lehrbuch der klinischen Diagnostik," G. Fischer.)

sphygmomanometer of this type consists of a mercury manometer, a rubber bag surrounded by an unyielding cuff for the arm and a rubber bulb or metal pump for forcing air into the arm bag. The bag should be at least 12 cm. wide (von Recklinghausen). The narrower armlet used by Riva-Rocci gave too high readings. The interior of the bag is connected with the manometer by a rubber tube. The outer portion of

the cuff should be made of leather or other unyielding material. There is an adjustable leak for lowering the pressure slowly.

Owing to their compactness, aneroid (i. e. without fluid) manometers are extensively employed in preference to the more bulky mercury manometers. The readings of an aneroid instrument should be frequently checked by comparison with a mercury manometer.

As furnished by some makers accuracy has been sacrificed to convenience by omitting the rigid outer cover of the cuff. The use of these instruments with an improperly made cuff should be discouraged.

Palpatory Method.—The maximum or systolic pressure can be determined by palpation of the radial artery. The cuff is snugly applied around the upper arm, but not so tight as to raise the pressure in the rubber bag. The armlet is then inflated until the pulse at the wrist is obliterated. The pressure is then slowly lowered by allowing air to escape through the small hole provided for this purpose. When the first pulse wave is felt in the radial the height of the mercury column is noted. This is taken as the systolic pressure. On slowly lowering the pressure below this point the pulse waves increase in size and after the pressure has fallen a certain distance, varying with the different individuals and with the same individual at different times, the size of the wave suddenly becomes smaller. This is considered the diastolic pressure. This palpatory method of determining the diastolic pressure is inexact and at the present time rarely employed. The systolic pressure is usually from 5 to 15 mm. lower than that obtained by the auscultatory method.

Auscultatory Method.—The bell of the stethoscope is placed over the brachial artery at the bend of the elbow two or three centimeters below the lower margin of the cuff. When the pressure in the cuff is raised above the systolic pressure the brachial artery is completely closed and no sound is heard. If the pressure is allowed to escape slowly a clear sound will be heard when the pressure in the artery above the cuff is sufficient to allow some blood to pass through the vessel. This is the systolic pressure. After the pressure in the cuff falls about 10 mm. of mercury, this sharp sound with each systole is replaced by a murmurish sound which is heard while the pressure falls about 15 mm. more. This is called the second phase. The murmurish character is probably due to a stenosis of the vessel produced at this stage. At a still lower pressure the murmur is changed to a clear but dull sound. This is the third phase, and lasts during a fall of about 20 mm. This sound increases in loudness for a time as the pressure continues to drop. At length the sound suddenly becomes weak and muffled. This is the fourth phase and is the point of diastolic pressure. About 5 mm. below this pressure the sound

disappears. This is designated the fifth phase. Some observers take the fifth phase as the diastolic pressure but this is a mistake. In aortic insufficiency frequently and in exophthalmic goitre occasionally the murmurish sound does not disappear until the mercury drops to zero.

The sounds are probably due to a water-hammer action. (Erlanger.) "(a) The first sound is heard at the instant the blood in the artery below the compression chamber shows a brusque acceleration with each pulse. (b) The intensity of the sound then increases through the second and third phases as long as the diminishing compressing pressure still suffices to occlude the artery during a part of diastole. It is during this stage that the energy liberated by the water-hammer presumably is constantly increasing. (c) The sudden dulling and weakening of sound (fourth phase) occurs exactly at the instant the compressing pressure leaves the artery open during diastole."

Careful study has shown that the auscultatory method of measuring both the systolic and diastolic pressure is trustworthy; the error not being more than 5 per cent.

The oscillometric and subjective sensation methods of sphygmomanometry are not in ordinary use, although the former is advocated by some Europeans, particularly with the application of Pachon's oscillometer.

Factors in the Accurate Determination of the Blood-Pressure.—The blood-pressure may be taken either in the sitting or recumbent position. It is important that the elbow should be at the level of the heart in order to avoid introducing an error from the hydrostatic pressure of the blood column. The cuff should be snugly applied to the arm with the distensible bag opposite the brachial artery. A small volume of air should remain within the bag when the manometer is at zero. The determination should be made as quickly as is consistent with accuracy. The first record should be disregarded as the pressure is practically always elevated above the usual level by the excitement or apprehension of having the pressure taken for the first time. It is well to make a series of observations whenever the blood-pressure is recorded. After each determination has been made the pressure should be allowed to drop to zero and there should be a sufficient interval before making the next record to permit the venous pressure (stasis) to fall to the normal level. (Norris.) It is a good rule to take consecutive readings until the blood-pressure has ceased to fall.

Normal Blood-Pressure.—The normal systolic blood-pressure is between 100 and 140 mm. Hg. The usual range is 120 to 140. The diastolic blood-pressure varies from 60 to 100. It is usually between 80 and 90. The blood-pressure increases with age. If the normal blood-

pressure at twenty years of age is considered to be 120 an increase of 1 mm. for every two years may be regarded as normal. (Faught.) This and similar rules have a very limited value as the pressure in healthy persons between the age of 50 and 70 is frequently between 115 and 130. It is lower in children than in adults and about 10 mm. lower in women than in men. It is increased by exercise and especially by emotion. The latter element is hard to eliminate. The diastolic pressure is less influenced by emotion than the systolic. There is a rhythmic variation with respiration; in most persons both systolic and diastolic pressure being higher during expiration than during inspiration. If the pressure in the cuff is allowed to fall slowly the first sound on auscultation will often be heard only during the expiration phase. After the mercury has fallen 3 to 5 mm. more a sound is heard with every beat. (Wiggers.) In some cases of chronic mediastinopericarditis and mediastinal tumor, when the flow of blood into the heart is checked in inspiration by pressure on the veins or by the dynamic condition, there is a marked drop in the arterial blood-pressure during inspiration accompanied by smaller pulse waves. This is due to diminished filling of the arteries. (Pulsus paradoxus.) A systolic pressure above 160 or below 90 and a diastolic pressure above 100 are certainly abnormal. Recent statistical study shows that systolic pressure above 140 for men, and 130 for women should be considered pathological. (Alvarez.) [1]

In the routine examination both the systolic and diastolic pressure should be determined. The difference between them is called the amplitude or pulse pressure. This normally varies between 35 and 50 mm.

Increased Blood-Pressure. Hypertonia.—In chronic nephritis and in essential hypertonia (hyperpiesis of Clifford Allbutt) the blood-pressure is greatly elevated. Systolic blood-pressures of 180, 200, 250, and even 300 or more occur. The diastolic pressure frequently rises to 120 or 130. These high pressures may be maintained for years. A high blood-pressure occurs when the flow through the smallest arteries and the capillaries is difficult owing to constriction of their calibre provided the left ventricle is sufficiently strong to force the blood through the narrowed vessels in the usual time. When the heart muscle weakens in these cases, the systolic blood-pressure frequently falls to 150 or less. This is an ominous sign. Increased constriction of the peripheral blood-vessels is unable to raise the blood-pressure in these cases. The whole circulation of blood will be slowed and there will be congestion in the lungs and veins. The deduction should not be drawn from these statements that

[1] Alvarez, *Arch. Int. Med., Chicago,* 1920, XXVI, 381.

a high systolic pressure indicates necessarily a strong heart. The heart may be so weakened as to produce marked symptoms of cardiac weakness such as dyspnea and edema and yet be able to maintain the high blood-pressure in cases of hypertension. As the heart progressively weakens in these cases the systolic pressure usually falls.

The blood-pressure will be little, if at all, elevated by increased and rapid cardiac action, if the finer arteries and the capillaries at the same time dilate. This explains the lack of elevation of blood-pressure with active exercise and severe muscular work. The velocity of the blood flow, however, increases greatly. Narrowing of the smaller arteries is usually due to contraction of their muscular wall which is under the control of the vasomotor nerves. Sclerosis of the blood-vessels in the absence of kidney disease does not raise the blood-pressure, except possibly when the splanchnic arteries are involved. Experimental constriction of the splanchnic arteries does not produce a permanent elevation of the blood-pressure. (Longcope and McClintock.)

A marked but transitory increase in the arterial pressure may occur in attacks of angina pectoris but it is not common. A temporary contraction of the arterioles with resulting elevation of the blood-pressure occurs in lead colic, in a shaking chill (rigor), as a result of injecting adrenalin and in some forms of acute nephritis. (F. Müller.) In congestive failure of the heart the blood-pressure is often elevated—the congestive hypertonia of Sahli. Hypertension also occurs in lead poisoning and in polycythemia vera.

In aortic insufficiency the systolic pressure is usually elevated. The diastolic pressure is lowered so that the pulse pressure in this disease is greatly increased. The systolic pressure in the leg is often much higher than in the brachial artery in aortic insufficiency. In aortic stenosis the pulse pressure is low. A transitory rise of blood-pressure due to emotion is well recognized. I have seen a marked elevation of both systolic and diastolic blood-pressure persistently present for months in cases of vasomotor neurosis. A subsequent fall to normal without evidence of cardiac or arterial disease confirmed the diagnosis.

The prognostic significance of an elevated blood-pressure is well shown by the statistical study of an American insurance company. Persons between the age of 40 and 60 with an average maximum blood-pressure of 142 mm. had a normal death rate. Those with an average pressure of 153 mm. showed a 30 per cent higher death rate, and those with a pressure of 161, a death rate 100 per cent above the normal.

Decreased Blood-Pressure. Hypotonia.—A systolic blood-pressure below 90 is considered pathological in an adult. In general, a per-

sistently low blood-pressure has little clinical significance, except as a diagnostic sign of Addison's disease. In this disease the systolic blood-pressure may fall to 50 mm. of mercury. In active pulmonary tuberculosis low blood-pressures occur, and they are also common in neurasthenic states. Hypotonia is said to be often met with in cases of visceroptosis, but probably it is due to the associated vasomotor disturbance. A very low blood-pressure is a characteristic sign of surgical shock.

In fever the diastolic blood-pressure is usually abnormally low because the outflow of blood through the arterioles and capillaries is facilitated by the dilatation of these vessels. The peripheral portion of the arterial system is widened as a result of a diminished tonus of the vasoconstrictors. The systolic pressure may remain high in fever if the heart is strong and as the diastolic pressure is low the pulse pressure will be increased. If the heart weakens the systolic pressure will fall and the pulse become small and soft.

Blood-Pressure in Auricular Fibrillation.—The blood-pressure determined by the usual methods gives us inaccurate results in auricular fibrillation, and other gross irregularities. Some recent writers state that they are valueless. This is not true because blood-pressure readings made in the ordinary manner enable one to recognize cases of hypertonia and this is important. In seven out of thirty-two cases of auricular fibrillation I found the systolic blood-pressure about 170 mm. In the small arteries the wide fluctuations of pulse pressure that occur in the brachial and radial arteries are not present, and Cohn and Lundsgaard [2] have devised a satisfactory method of obtaining the pressure in the digital artery of the ring finger. The technic devised by Gaertner is employed. In normal persons the figures obtained by this method are 10 to 20 mm. lower than the pressure in the brachial artery. The method has not however been adopted clinically nor is there need of such adoption.

Venous Pressure

The accurate determination of the venous pressure has not been shown to have definite clinical value. (Wiggers). The normal pressure at the level of the heart varies from 3 to 15 cm. of water. It is elevated in some pathological conditions.

Method of Gaertner: The subject sits upright before the measuring apparatus and the level of the costosternal angle is ascertained. The relaxed arm bending the elbow at 120 degrees is then supported at the elbow

[2] Cohn and Lundsgaard, *J. Exper. M., N. Y.*, 1918, XXVII, 487.

and the hand by the observer. The whole arm is raised until the veins on the back of the hand collapse. The distance between this level and that of the center of the costosternal angle represents the auricular pressure in centimeters of water. A modification suitable for use at the bedside has been suggested by von Recklinghausen. One hand of the patient is placed by his side as he lies in bed, the other on his thigh. If the veins of the hand on the thigh collapse, while those of the one on the bed fill, the venous pressure is normal. If the veins of both collapse the pressure is low; if the veins of both hands fill, it is high. The veins may be so small due to increased tonus that this test cannot be made.

A variety of instruments for measuring venous pressures have been invented. The best are those of Hooker,[3] for indirect measurement and Moritz and Tabora,[4] for direct measurement. The latter procedure consists of inserting a needle connected with a capillary manometer directly into the median vein of the forearm. This is probably the most accurate method.

Capillary Pressure

Measurement of the pressure in the capillaries promises to be of value in clinical diagnosis. Capillary microtonometers have been devised by Lombard,[5] Danzer and Hooker [6] and others. Pressures between 10 and 25 mm. of mercury are obtained normally but the pressure varies in different capillaries in any area examined. Boas-Frant [7] found that cases of arterial hypertension could be divided into two groups in one of which the capillary pressure was elevated and in the other it was normal. The elevated capillary pressures ranged between 25 and 70 mm. In essential hypertension capillary pressure was low.

GRAPHIC METHODS

The Sphygmogram

The sphygmogram is a graphic representation of the variations of pressure within an artery. It is a blood-pressure curve but the scale on which the ordinates are drawn remains unknown. (v. Frey.)

[3] Hooker, *Am. J. Physiol., Bost.,* 1914, XXXV, 73.
[4] Moritz and Tabora, *Deutsches Arch. f. klin. Med., Leipz.,* 1909, XCVIII, 475.
[5] Lombard, *Am. J. Physiol., Bost.,* 1912, XXIX, 335.
[6] Danzer and Hooker, *Am. J. Physiol., Bost.,* 1920, LII, 136.
[7] Boas-Frant, *Arch. Int. Med., Chicago,* 1922, XXX, 40.

The sphygmograph was introduced into clinical medicine sixty years ago by Marey the French physiologist. For many years it proved of little or no real service in the clinical study of disease. Gradually its use was abandoned. Less than twenty years ago, it was called a fascinating little toy by the writer of a well-known textbook on physical diagnosis. But even before these words of condemnation were written two men working quietly and independently had found that this instrument properly used gave valuable information. It took years to convince the medical world that this was true. Mackenzie [8] or Burnley, England, showed in a paper published as early as 1893 that tracings of the radial pulse enabled him to time events in the cardiac cycle when employed in the investigation of the venous pulse. A few years later Wenckebach [9] a young physician in Holland was able by keen analysis to interpret correctly cardiac irregularities from radial tracings alone. The work of these men with the despised and rejected sphygmograph was the founda-

FIG. 44.—Sphygmogram of the radial pulse. The space *E* is the period of ventricular systole when the aortic valves are open; the space *G* the period of ventricular diastole; *s* is the pulse wave due to the ventricular systole; *n* the aortic notch, *d* the dicrotic wave; and *p* wave due to instrumental defect. (Mackenzie, "The Study of the Pulse," Young J. Pentland.)

tion on which our present knowledge of disorders of the cardiac rhythm has been built.

All the earlier syphgmographs possessed serious instrumental defects which distorted the size and shape of the pulse. The tracings do, however, reveal the time of events occurring during the cardiac cycle. In Fig. 44 is a tracing of the radial pulse with the old Dudgeon sphygmograph. The elevation *d* on the descending limb is called the dicrotic wave. It is important as it marks the time that the aortic valves close. The space *E* in the tracing is the period when the aortic valves are open and the ventricle pours its contents into the aorta. The space *G* is called the period of diastole although Mackenzie recognized that it included the early part of systole (isometric) when the aortic valves are closed. The rise of the radial pulse wave does not occur at the instant the aortic

[8] Mackenzie, *J. Path. & Bacteriol.*, 1894, I, 53.
[9] Wenckebach, *Ztschr. f. klin. Med., Berl.*, 1898, XXXVI, 181.

valves open as it takes one tenth of a second for the wave to travel from the aorta to the wrist. This delay is well shown in simultaneous tracings of the carotid and radial pulse, Fig. 45, and more strikingly in Fig. 46.

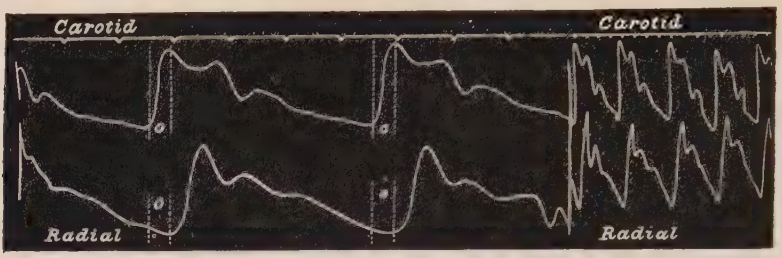


FIG. 45.—Simultaneous tracings of the carotid and radial pulses from a case of aortic regurgitation. The first portion of the tracing was taken when the paper was moving at the more rapid speed. The space (o) representing the radial-carotid interval is nearly 0.1 of a second. (Mackenzie, "The Study of the Pulse," Young J. Pentland.)

The alternation of large and small waves, the pulsus alternans, can be recognized in tracings made with the ordinary sphygmographs. It was discovered and named by Traube in 1872. The importance of this sign

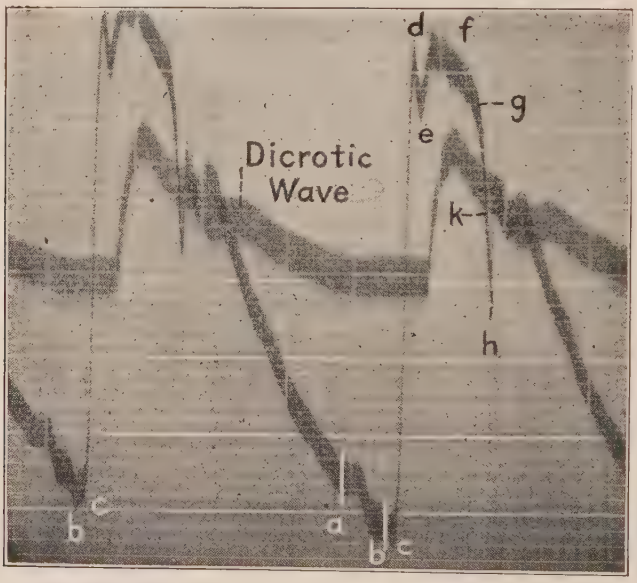


FIG. 46.—Tracing made by the optical method showing the difference between the subclavian (central) and radial (peripheral) pulse in man. The delay of the radial is well shown. Preliminary vibration due to auricular systole *a–b*. Vibration due to isometric rise of tension in the ventricle *b–c*. Opening of semilunar valves *c*. End of systole *g*. Incisura of central pulse *h*. (Wiggers, "Modern Aspects of the Circulation in Health and Disease," Lea and Febiger.)

of serious impairment of the power of contractility justifies the routine employment of the sphygmograph in every case of evident or suspected cardiac weakness in elderly people.

Different Types of Sphygmographs.—The von Frey, the Dudgeon

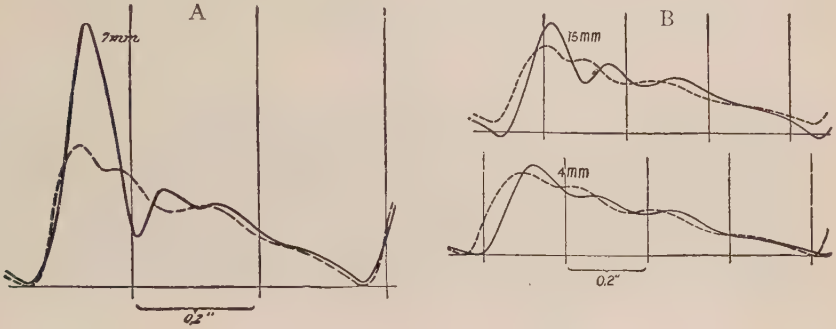


FIG. 47.—Experimental tests of the efficiency of sphygmographs. Types of tracings. A, Dudgeon record; B, Jacquet records. Both tests compared with true curves (broken line). The curve obtained with the Dudgeon apparatus rises from the base line at the same time as the true curve and the two return to the base line simultaneously. Hence for time relations the Dudgeon is sufficiently exact. The height and contour of the wave is incorrectly reproduced. The dicrotic wave is too low. The curves obtained with the Jacquet are even worse. The rise and return to the base line are delayed. The dicrotic wave is not recorded but is replaced by a series of after-vibrations which yield a wave often mistaken for the dicrotic wave. (After Petter, Wiggers, "Modern Aspects of Circulation in Health and Disease," Lea and Febiger.)

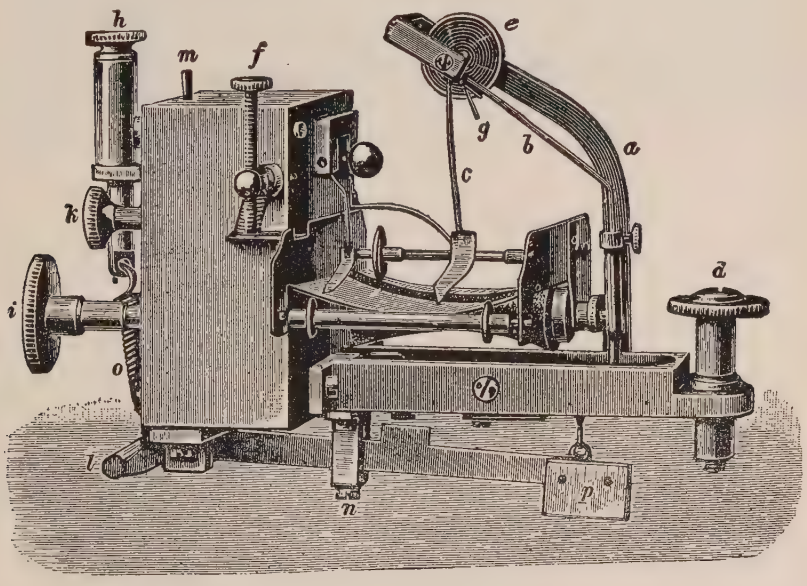


FIG. 48.—Sphygmograph of Frank and Petter. (Edens, "Lehrbuch der Perkussion und Auskultation," J. Springer.)

and the Jacquet are wrist sphygmographs and have been largely used. All distort considerably the true pulse curve, see Fig. 47. In 1908 Frank and Petter devised their sphygmograph in which the technical deficiencies of the earlier instruments have been removed. It gives a true tracing of the size and shape of the arterial pulse. This instrument is shown in Fig. 48 and excellent tracings made with it in Fig. 49. In studying

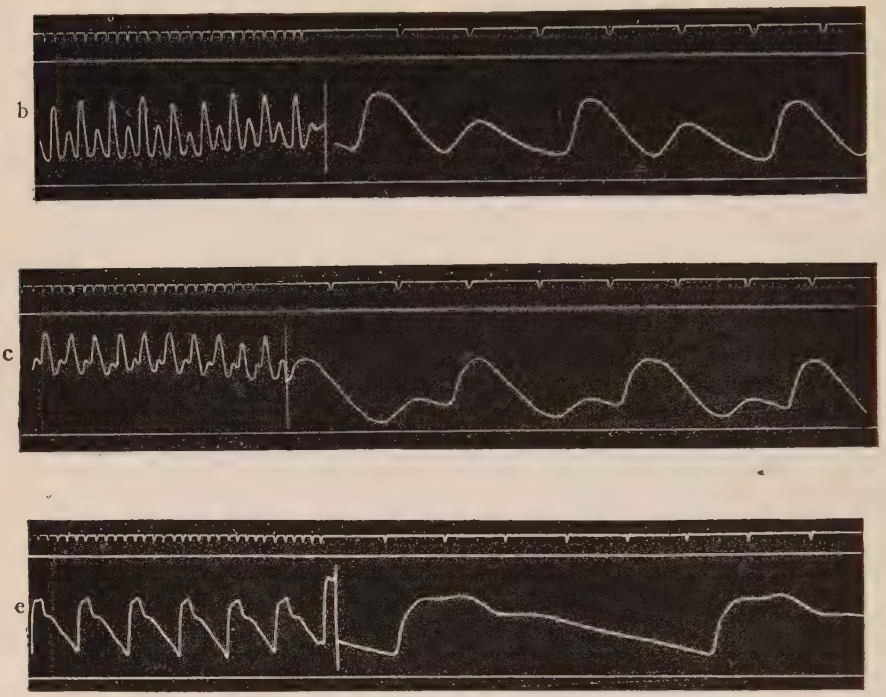


Fig. 49.—*b*, dicrotic pulse; *c*, hyperdicrotic pulse; *e*, pulse in aortic stenosis. (Edens, "Lehrbuch der Perkussion und Auskultation," J. Springer.)

pulse irregularities it is very helpful to make simultaneous tracings of the radial and jugular pulse, or of the cardiac apex and the jugular. Jacquet devised a sphygmocardiograph which has met with favor. It is a compact, finely made instrument which in addition to the radial tracings permits the registration of two additional curves (jugular, apex-beat or carotid). The tracings are made on strips of smoked paper of relatively short length. For clinical use the ink writing polygraph of Mackenzie has many advantages. See Fig. 50. Long tracings can be made without renewing the roll of paper. But on the other hand the vibration period of the writing levers is so slow that true curves are not obtained. The radial tambour and lever are especially deficient in ability to reproduce

the details of the pulse, as they have a vibration frequency of 4.5 per second (Wiggers). The vibration frequency required to reproduce the true pulse curve is 32 per second. The Frank-Petter apparatus alone has this vibration rate.

Instead of using ponderable levers writing on paper, registration by photographic methods of a beam of light has been extensively employed in recent physiological and clinical studies. Frank devised a segment capsule for optical registration. On a thin rubber sheet that is stretched over the surface of the capsule a tiny mirror is fastened that reflects a beam of light to a moving photographic plate or film. Wiggers and Baker have recently devised an improved form of sphygmograph for use with

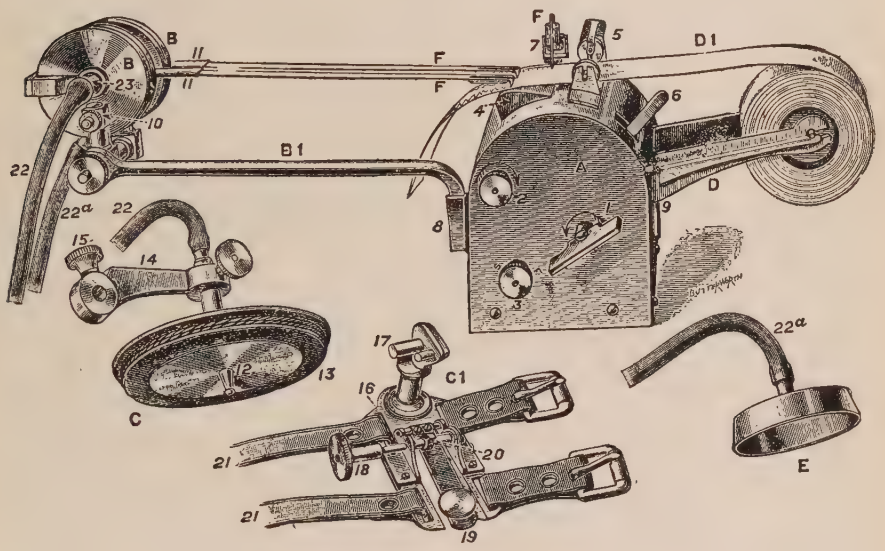


Fig. 50.—Ink polygraph. (Mackenzie, "Diseases of the Heart," Oxford University Press.)

Frank's segment capsule. A tracing of the subclavian and radial pulse made by the optical method is shown in Fig. 46.

The Central Pulse.—The pulse in the aorta, subclavian, and lower part of the carotid arteries is called the *central pulse*. It differs greatly in its form from that of the *peripheral pulse* which is found in the radial arteries. This change is due first, to the inability of the arteries to transmit to the periphery the pressure changes occurring in the aorta, owing to the low vibration rate of their walls, and, second, to the reflection of waves from the periphery to the central arteries.

The difference between the central and peripheral pulse is clearly seen in Fig. 46.

Form of the Central Pulse.—After small preliminary oscillations due to the isometric rise of tension in the ventricle (b–c) Fig. 46, there is a sharp and high rise in the wave marking the beginning of the ejection period, the first systolic wave, (c–d–e). The pressure then continues high, forming a more or less definite summit; this is the systolic accessory wave, (e–f). The wave then falls slightly during the remainder of systole. With the closure of the semilunar valves there is a sudden and rapid drop (g–h). This forms the incisura (h) of the central pulse. Immediately following this are the after-vibrations of the semilunar valves and the blood column in the aorta (k). From this point onward there is a steady fall in the curve during diastole due to the flow of blood through the smaller arteries and capillaries and the consequent drop in pressure. At the end of diastole there is a small wave (a–b) due to the auricular contraction.

The Peripheral Pulse.—The character of the pulse as exhibited in the radial artery is so modified in transmission and by peripheral influences that its resemblance to the central pulse is slight. The systolic rise is more gradual but the first systolic wave is more prominent and usually forms the highest elevation of the pulse curve. The summit is more rounded and the amplitude is less. The systolic accessory wave is usually not as distinct as in the central pulse. It can disappear entirely from the descending limb if the flow of blood to the periphery is increased as in the fever pulse. If on the contrary the flow of blood to the periphery is impeded by contraction of the vessels or arteriosclerotic narrowing of the smaller arteries, then the systolic accessory wave is more prominent in the radial tracing. It becomes broader and higher and can even exceed the height of the first systolic wave. Such a pulse curve is termed anacrotic.

The incisura of the central pulse is replaced by the dicrotic notch and wave. The origin of the dicrotic wave is still unsettled. It often occurs a little later than the closure of the semilunar valves, but no considerable error arises from marking diastole as beginning with the dicrotic wave on the radial tracing. It is at least in part due to vibrations reflected from the periphery. The ascending portion of the pulse wave is called the anacrotic limb, and the descending portion the catacrotic limb.

The Dicrotic Pulse.—The dicrotic wave is larger when the pulse is soft, and can then be distinctly felt by the palpating finger as a second wave. The pulse then beats double. It is dicrotic. Dicrotism occurs chiefly in fever. With increasing fever and decreasing arterial tension

the pulse is first hypodicrotic, then completely dicrotic and finally with very rapid pulse-rates it becomes hyperdicrotic. A dicrotic pulse is one in which the dicrotic wave is found almost on the same level as that of the primary systolic wave. See *b*, Fig. 49. If the dicrotic wave is at a higher level it is hyperdicrotic. See *c*, Fig. 49. In high fever when the pulse waves follow each other with great rapidity the dicrotic wave may be absent (monocrotic pulse).

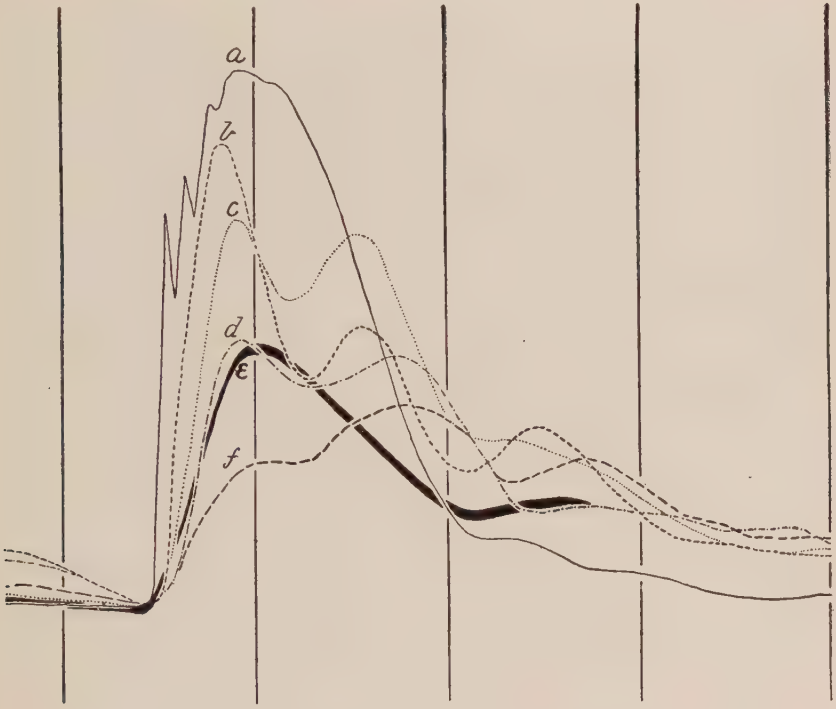


FIG. 51.—Curves illustrating the chief types in cases of aortic disease and a normal pulse curve. The normal pulse record is drawn with a heavy line. The vertical lines represent time in fifths of a second. (a) Class I: Abrupt up-stroke and thrill. (b) Class III: Relatively abrupt up-stroke with two peaks, the second appreciably smaller. (c) (d) Class IV: Less abrupt up-stroke with two equal peaks (bisferiens). (e) Normal record. (f) Class V: Anacrotic pulse. (Feil and Gilder, Heart, Lond., VIII, Shaw and Sons.)

Pulsus Tardus in Aortic Stenosis.—The ascending limb often shows a depression or a bulging and the systolic accessory wave is higher than the systolic primary wave, see *e*, Fig. 49. The pulse wave presents a low appearance and the diastolic portion—the catacrotic limb—forms a long gradual descent.

Pulsus Celer in Aortic Insufficiency.—(The collapsing or water-

hammer pulse). The pulse is very high and the primary wave rises abruptly. The descending limb is steep. The rapid decline indicates that the diastolic pressure is low compared with the systolic pressure. The various modifications of the radial pulse in aortic insufficiency as recorded by an optical sphygmograph are shown in Fig. 51. Two prominent summits may be present (pulsus bisferiens). The impression of the water-hammer quality is due to the abruptness with which the pressure rises and not to the "collapse" of the pulse. (Feil and Gilder.)

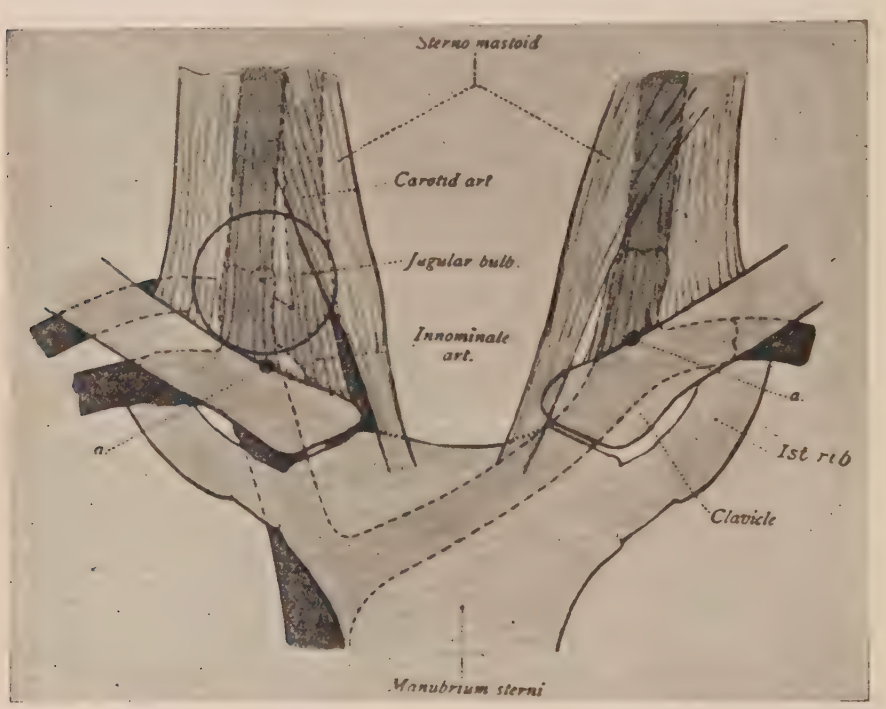


Fig. 52.—Diagram showing the situation of the carotid artery and the jugular vein. (Cowan, "Diseases of the Heart," Edward Arnold and Company.)

More important than the study of shape and rate of the arterial sphygmogram is the study of the rhythm so ably developed by Wenckebach with his principle of the dominant rhythm.

THE PHLEBOGRAM (THE VENOUS PULSE)

For making tracings of the jugular pulse the Mackenzie and the Jacquet polygraphs are largely used. Receivers are placed over the pulsating veins and by air transmission through rubber tubes the movements are

conveyed to tambours with attached writing levers. More accurate records can be made with the Frank capsule by reflecting a beam of light to a moving photographic plate. The ordinary polygraph gives records that are of great value in the diagnosis of cardiac irregularities. The instruments are compact and can readily be used by the physician at the bedside or in the consulting room. Optical tracings require elaborate apparatus and cannot be employed outside of a hospital. They have been chiefly of value in physiological investigations, and have yielded little or no definite information of clinical importance that cannot be obtained with simple tambours by air transmission.

The pulsation is best recorded over the jugular bulb. This is situated just above the inner portion of the right clavicle between the two heads of the sternomastoid muscle. (Fig. 52.) The patient should be in the recumbent position with the head supported by a single pillow. The muscles should be relaxed by dropping the chin and turning the head a little to the right.

The Jugular Pulse.—A tracing of the jugular pulse can be made from a good proportion of normal persons. The jugular tracing is not a record of changes of venous pressure but the record of the filling of the veins; that is, a volume pulse. (Wenckebach.) The jugular is like a rapid flowing stream in a narrow bed. Damming back of the onward flow causes the level of the stream to rise rapidly. The sudden distention of the walls of the jugular when the current is blocked gives rise to wave formation. This is shown in a simple experiment described by Wenckebach. Sudden, firm pressure over a full jugular vein by a finger placed just above the clavicle causes a wave to ascend the vein, which is not only well seen in a tracing but by the naked eye. This is followed by a sudden emptying of the vein, indicated by the sharp drop in the tracing, when the pressure is released. The rapid escape of blood accumulated in the jugular fills the large veins below and their distention with blood checks the onward flow in the jugular. As a result this vessel is quickly filled with blood from above, causing a second smaller wave. In actual tracings of the jugular pulse this second small wave is rarely seen owing to the ventricular contraction. In the cardiac cycle are two checks to the onward movement of the blood in the jugular veins,—the first during auricular systole and the second in the first phase of ventricular diastole. These sudden physiological arrests of the venous blood-flow produce waves in the jugular in a similar manner to those produced artificially in the experiment just described.

The Negative or Physiological Venous Pulse.—There are three waves in the normal jugular pulse. See Fig. 53. The first or *a* wave

is due to the auricular systole. It is the rise of the pressure in the auricle during systole that dams back the blood in the big veins causing them to swell. This sudden increase in volume of the jugular forms the *a* wave. The wave falls with the drop of pressure in the auricle occurring in the latter part of auricular systole, the so-called inflow phase when blood again flows into the auricle. The second or *c* wave is due partly to the impact of the carotid on the distended jugular and partly to change in intra-auricular pressure with ventricular systole. It is syn-

FIG. 53.—Simultaneous tracings of the jugular and radial and carotid and radial pulses. *E* and *E'*, duration of ventricular outflow, right and left, through arterial orifices; *a*, auricular wave; *v*, ventricular wave; *c*, carotid wave; *x*, auricular depression; *y*, ventricular depression. The dotted lines between *a* and *x* indicate probably the true venous pulse wave. (Mackenzie, "The Study of the Pulse." Young J. Pentland.)

chronous with the carotid pulse and often its form resembles that of the carotid pulse especially when the receiving tambour is placed near the carotid artery. The *c* wave is perhaps sometimes increased by the pressure of the aorta on the right auricle (Wenckebach). The cause of the third or *v* wave is not definitely known. It is probably due to venous stasis during ventricular systole. The time of its onset may vary, but usually its sharp rise occurs in early ventricular diastole. The abrupt fall from its summit marks the opening of the auriculartventricular valves. This is the most definite point of time in the cardiac cycle recorded in the venous tracing. (Mackenzie.)

In addition to the three waves there are two depressions, or negative waves, *x* and *y*, in the venous pulse. The first, *x*, occurs after *a* and is caused by the flow of blood into the auricle during the latter part of auricular systole, the inflow phase, and the first part of auricular diastole. The ventricle in its contraction pulls down the auriculoventricular region and this movement aids in the filling of the auricle and accelerates the onward flow of blood from the jugular into the superior vena cava. The blood that has been dammed back in the auricle and great veins during the ventricular systole flows into the ventricle when the tricuspid valve opens and this causes the negative *y* wave in the phlebogram.

Occasionally when the diastole is long a fourth positive wave is seen in venous tracings. This has been named the *h* wave by Hirschfelder and the stasis or *s* wave by Rihl. Its cause is unknown.

The Positive or Pathological Venous Pulse.—The *v* is larger and begins in ventricular systole. The *c* wave may be lost in the large *v* wave. This type of pulse occurs in tricuspid regurgitation. It is almost always found in association with auricular fibrillation. Very rarely is the positive form of venous pulse seen when the heart rhythm is normal. Wenckebach stated in 1914 that he had observed only three cases.

The chief clinical importance of tracings of the venous pulse is the

FIG. 54.—Simultaneous tracings of the liver pulse and carotid pulse. The liver pulse is of the auricular form, the fall after the auricular wave *a* begins before the carotid pulse. (Mackenzie, "The Study of the Pulse," Young J. Pentland.)

information it gives of the presence of the auricular systole and the time of its occurrence in the cardiac cycle. The a–c interval as measured in jugular tracings is normally one-fifth of a second. Prolongation of this interval is evidence of the first stage of heart-block. Simultaneous trac-

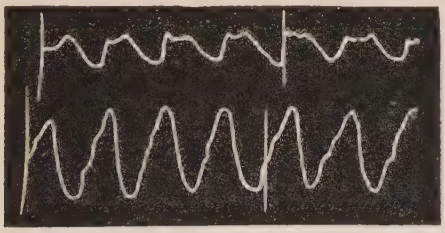


FIG. 55.—Simultaneous tracings of the carotid and liver pulse of the ventricular form, showing the rise in the liver tracing to occur with the carotid pulse. The upper tracing is the carotid, the lower, the liver. The large single wave in the liver tracing occurs in ventricular systole, hence it is a *v* wave. (Mackenzie, "The Study of the Pulse," Young J. Pentland.)

ings of the radial and jugular pulse enable the physician to diagnose the different forms of heart-block, to distinguish whether extrasystoles originate in the auricle or ventricle, and to diagnose definitely auricular fibrillation by the absence of *a* waves.

The liver pulse is a form of the venous pulse. It usually shows no arterial *c* wave. Rarely one is seen due to a pulsation transmitted from the underlying abdominal aorta. Both a negative and positive form of liver pulse occur. They have the same significance in the liver as in the jugular pulse. The excellent tracings of the liver pulse shown in Figs. 54 and 55 are taken from Mackenzie's "Study of the Pulse."

THE ELECTROCARDIOGRAM

The invention of the electrocardiograph twenty years ago has made possible a great advance in the knowledge of the cardiac mechanism and in the clinical diagnosis of the cardiac irregularities. It had been known for many years that the contraction of a muscle generated a weak electrical current. Over sixty years ago it was demonstrated that at each contraction of the heart electrical currents were developed. (Kölliker and Müller.) Waller [10] in 1887, studied the action currents of the human heart by means of the capillary electrometer.

In 1903 Einthoven,[11] a Dutch physiologist, constructed the string galvanometer. It is this instrument that is now used as the electrocardiograph in clinical laboratories. It consists essentially of a very delicate fibre of silvered glass or quartz, suspended between the poles of a powerful electromagnet. Each end of the fibre is connected to an electrode applied to an arm or leg. From the surface of the body the active current of the heart is thus conducted through the fibre which becomes deflected to one side or the other according to the direction of the current. The string galvanometer is based upon the principle "that when a conductor carrying an electric current is placed at right angles to a magnetic field the conductor moves in a direction at right angles to both the field and the current." (Wiggers.) Thus in Fig. 56 if a minute current passes down the fibre GS, as it lies in the magnetic field, the fibre moves in the direction indicated by the central arrow in a plane at right angles to the lines of magnetic force across the gap. The magnified shadow of the fibre moves across the horizontal slit of a camera and the movements are registered upon a photographic plate or film.

The action current of the heart is the sum product of the multitude of the action currents of all the muscle fibres of the heart. In the heart as

[10] Waller, *J. Physiol.*, 1887, VIII, 229.

[11] Einthoven, *Ann. d. Physik, Leipz.*, 1903, 4 F., XII, 1059. *Arch. f. d. ges. Physiol.*, 1909, CXXX, 287.

in every muscle the stimulated parts are electronegative to the passive parts.

The form of the electrocardiogram differs in the same subject according as the electrodes are applied to one or other part of the body surface. The following standard leads are used in routine diagnostic work:

Lead I	Right hand and left hand
Lead II	Right hand and left foot
Lead III	Left hand and left foot

The normal electrocardiogram of the heartbeat consists of a series of waves or deflections. They have been named by Einthoven *P*, *Q*, *R*, *S*,

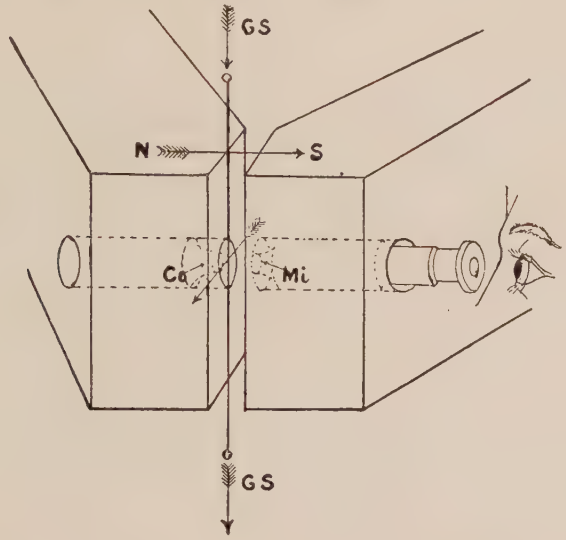


FIG. 56.—A diagram illustrating the construction of the string galvanometer. The poles of the magnet (N, S) are seen in outline; the recording fibre (GS) lies vertically between them; its movements may be observed through a microscope (Mi). The movements of the string are in a plane parallel to the faces of the magnetic poles, as indicated by the central arrow. (Co) is a condenser. (Lewis, "Clinical Electrocardiography," Shaw and Sons.)

and *T*. Each upward deflection indicates predominant negativity (activity) at the base or right side of the heart; each downward deflection signifies predominant negativity (activity) at the apex or left side of the heart. (Fig. 57.)

The first deflection *P* represents the auricular contraction. It is a rounded summit in health. In some pathological conditions the *P* wave although monophasic is directed downwards. During the interval between the end of *P* and the beginning of the initial ventricular deflection

(Q or R) the fibre is at rest while the impulse is being conducted along the auriculoventricular bundle from the auricles to the ventricles. The

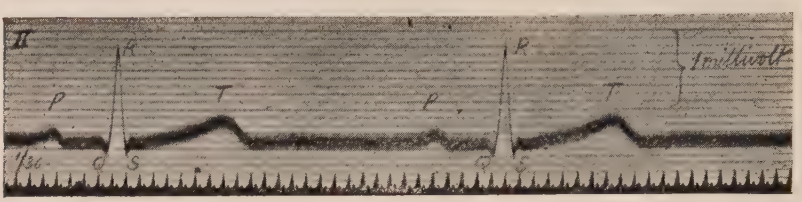


FIG. 57.—An electrocardiogram from a normal subject, showing the auricular summit (P) and the ventricular deflections (Q, R, S and T). The lead is marked in the upper left-hand corner; the time, at the bottom, is in one-thirtieth second, the ordinates each represent one-tenth millivolt. (Lewis, "Clinical Electrocardiography," Shaw and Sons.)

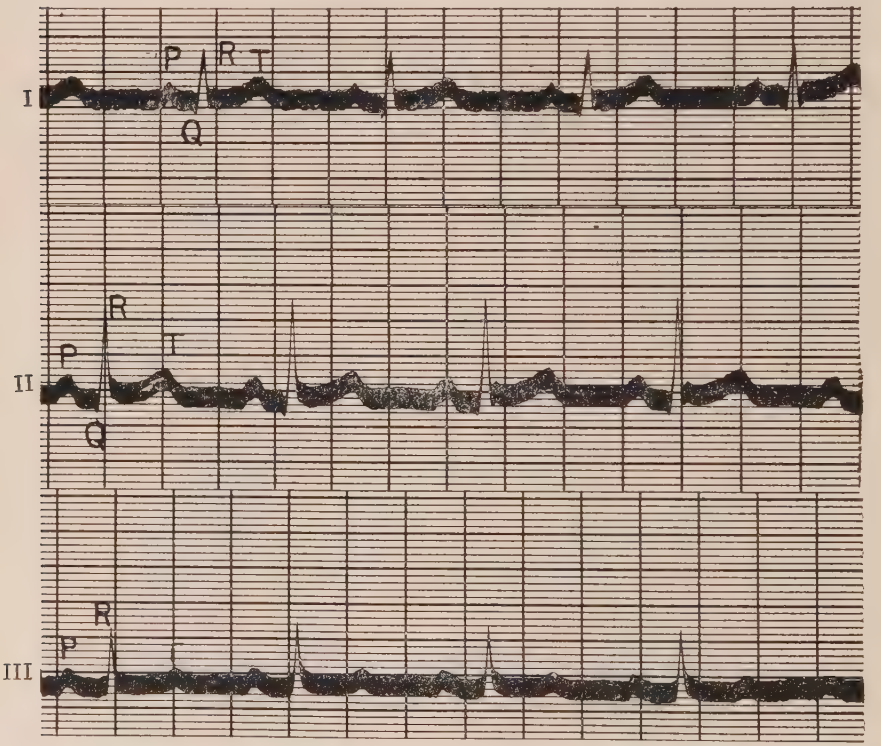


FIG. 58.—A normal electrocardiogram in the three leads, showing the auricular summit P and the ventricular deflections Q, R and T. Note that the type of curve changes with the lead and R is tallest in Lead II. The time indicated by vertical lines is in one-fifth second. (Dr. Paul D. White.)

ventricular complex varies in the number of its deflections. It may commence with a small deflection Q or this may be absent in all leads. R, a

steep tall spike, is constantly present. The curve may return to the base line or pass below forming *S*, a steep depression of variable depth. Toward the end of the ventricular complex is the smaller *T* wave which forms a broad rounded summit. The deflections *P* and *R* are not synchronous with the contractions of the auricles and ventricles, but precede them by a short space of time.

The value of the electrocardiogram can be summarized as follows: (1) The presence and time of the auricular contractions can be more definitely determined than in venous pulse tracings. Furthermore in many subjects a venous pulse is not present. (2) The site of origin of extrasystoles can be located. Not only can auricular and ventricular extra-

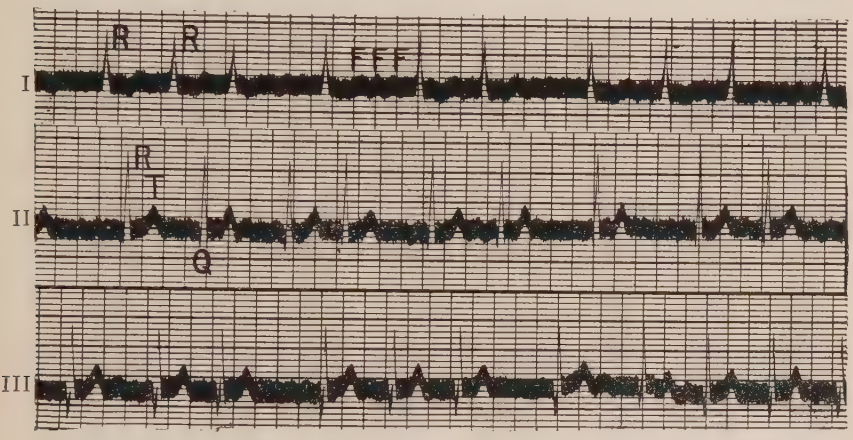


FIG. 59.—A curve of auricular fibrillation. Note that the ventricular complexes are placed at irregular intervals, and that the *R* summits vary in height. There are no P summits. The coarse oscillations especially well seen in Lead I during diastole are due to the fibrillations of the auricles. (Dr. Paul D. White.)

systoles be distinguished by characteristic changes in the curve but it is possible to tell whether extrasystoles arise in the left and apical or the right and basal part of the heart. (3) A positive diagnosis of auricular flutter can only be made with the electrocardiogram. (4) The presence of aberrations in the ventricular complex is evidence of myocardial degeneration or inflammation recognizable in no other way. (5) It is the only method of recognizing preponderant hypertrophy although it frequently fails to be a satisfactory one. (Herrman and Wilson, Cohn.) (6) Digitalization of a patient, hypothyroidism and at times myocardial exhaustion are indicated by flattening or inversion of the *T* wave in Lead II.

In auricular fibrillation the *P* deflection is absent. (Fig. 59.) Be-

tween the ventricular complexes of normal contour in auricular fibrillation fine or coarse irregular undulations occur. Auricular flutter is characterized by definite small waves recurring regularly 200 to 400 times per minute. The ventricular complexes may occur regularly or irregularly but always with a definite relation to the auricular waves. A 2 : 1 rate is common. The *P* and *T* waves may merge.

The initial deflection *R* in most cases of aortic insufficiency and other hypertrophies of the left ventricle is abnormally high (Leads I and II)

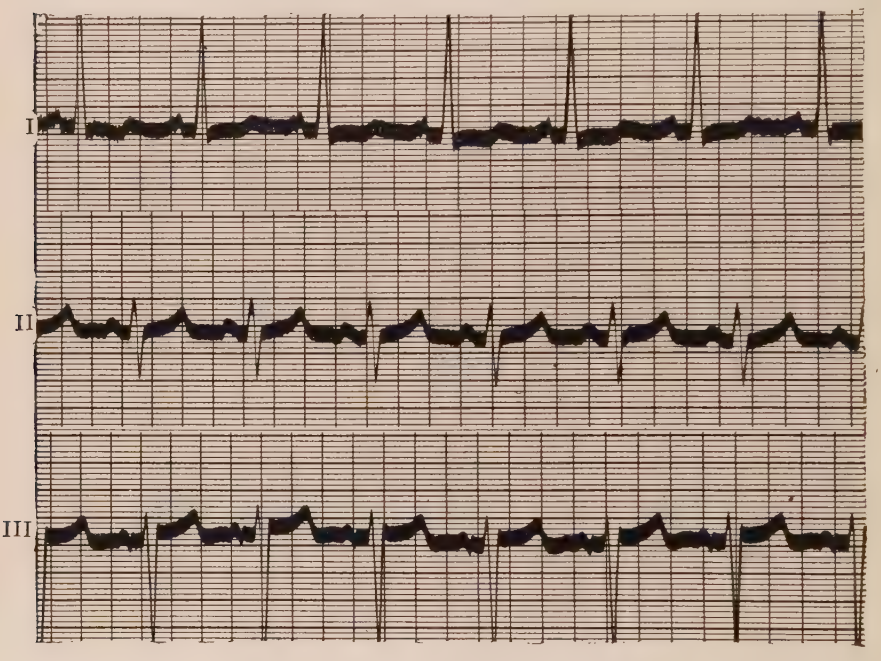


Fig. 60.—Left axis deviation (left ventricular preponderance). Note that *R* is tallest in Lead I and that *S* in Lead III is very deep. (Dr. Paul D. White.)

while the *S* is deep in Lead III. (Fig. 60.) On the contrary in preponderating hypertrophy of the right ventricle the *S* wave is abnormally deep in Lead I and *R* is high in Lead III. (Fig. 61.) The electrocardiograpic diagnosis of hypertrophy has been repeatedly called in question. These changes in the form of the curve are evidence of ventricular preponderance rather than direct indication of hypertrophy of one or the other ventricle. They are of considerable value in clinical diagnosis but must be interpreted with caution, as curves of preponderance may be obtained from a normal heart when it occupies an abnormal position in the

chest. (Cohn.) In cases of mitral stenosis the *P* wave is usually enlarged. This is ascribed to auricular hypertrophy. The size and position of the *T* wave are of some value as an index of the state of the heart muscle. A large *T* wave is regarded as a sign of vigorous heart action. A negative wave may occur normally in Lead III, but not in Leads I and II. The inversion may be temporary, and is produced regularly by digitalis, forming an early and trustworthy indication of digitalis action on the heart. (Cohn.) The presence of an inverted *T* wave in Leads I and II in a patient who has not been taking digitalis indicates hypothyroidism

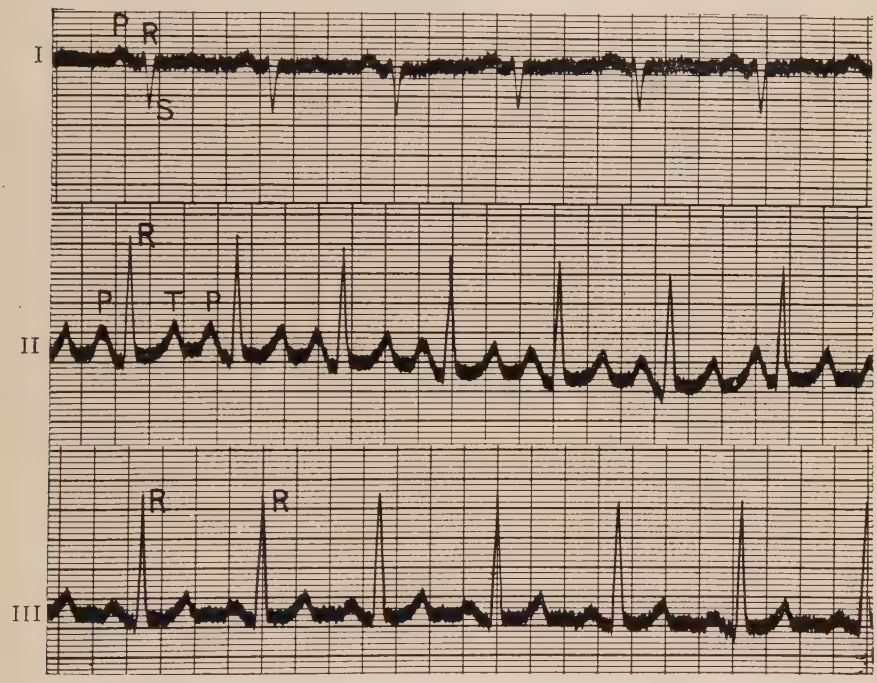


Fig. 61.—Right axis deviation (right ventricular preponderance). R in Lead I is almost absent, while *S* is prominent. In the third lead *R* is very tall. Note the high *P* waves in the second lead. (Dr. Paul D. White.)

or a badly damaged myocardium. Sixty-five per cent of the cases in a series studied by Willius died in 12.7 months.

Isolated ventricular extrasystoles change the electrocardiogram in characteristic fashion. The deflections are strikingly typical. The waves are of large amplitude and broad base and are followed by *T* waves of opposite potential. If an extrasystole arises in the wall of the right ventricle the initial deflection is a high upright spike in Lead III and usually in

Lead II. See Fig. 62. An extrasystole that has its origin in the left ventricle begins in Lead III with a large downward deflection.

Auricular extrasystoles are characterized by a premature occurrence of the entire group of *PRT* waves. The *P* wave is not exactly like a normal *P* wave unless its origin is very near the sinus node. When the extrasystole arises in the lower part of the auricle the *P* wave is inverted. (Fig. 63.) The ventricular complexes following the premature *P* wave are usually normal. When an extrasystole arises in the auriculoventricu-

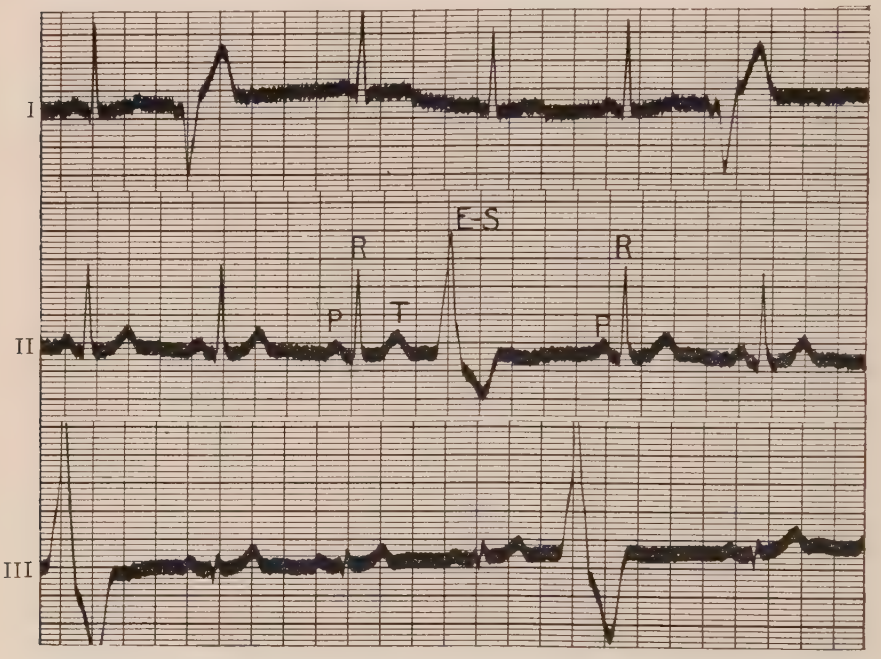


Fig. 62.—Ventricular extrasystole. The form of the premature ventricular complexes indicates that they arose in the right and basal portions of the ventricle. (Dr. Paul D. White.)

lar node the *P* wave is either closely followed by the *R* wave of the ventricular complex or the *P* wave occurs within the ventricular complex. If the point of origin is in His's bundle the *R* and *T* deflections are normal and the extrasystole is identified by its premature occurrence and the absence of a *P* wave at the proper interval before the ventricular extrasystole. The curve generally shows an undisturbed auricular rhythm. The presence of auricular systoles at regular distances indicates that the premature beats have arisen below the auricle. As the ventricular complexes are normal the extrasystoles must have had their origin above the

main division of His's bundle. Hence the focus of abnormal impulse formation must lie in the stem of the bundle.

THE ORTHODIAGRAM AND THE TELERADIOGRAM

The utilization of radiology has proved of great value in the diagnosis of disease of the heart and aorta. The size of the heart can be determined by orthodiagraphy or teleradiography with a certainty that is impossible

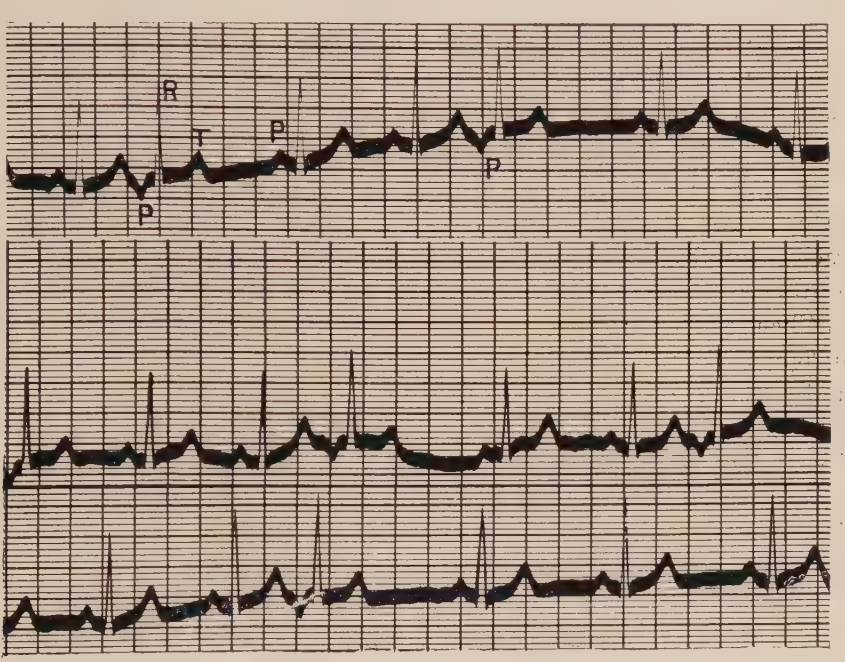


Fig. 63.—Extrasystoles of auricular origin. Note that the premature auricular complexes are inverted. (Dr. Paul D. White.)

by means of percussion. Changes in the form and position of the heart that are characteristic of different pathological conditions are revealed only by the Roentgen ray. In the early recognition of syphilitic aortitis and of aneurism fluoroscopy of the aorta has been of great service.

Simple fluoroscopy and plates made with the patient near the x-ray tube show changes in the shape and position of the heart and little else. Owing to the divergence of the rays from the anticathode the picture of the heart is so magnified and distorted on the screen and plate that only great enlargements of the heart can be definitely recognized. The at-

tempt to diagnose a slight or moderate increase in the size of the heart from ordinary plates has been a fruitful source of error.

Fortunately means have been devised for utilizing the Roentgen rays so that the size of the heart by sagittal transillumination of the chest can be accurately determined. Orthodiagraphy is a method brought into use by Moritz for measuring the heart in various diameters. He invented an apparatus called the orthodiagraph for this purpose. Only parallel rays of light are utilized. The tube and a small fluoroscopic screen are so united that the anticathode and the screen while freely movable are always in alignment. A small diaphragm shuts out all but the central beam of light, so that the central rays pass from the tube through the center of the screen in a straight line to the eye of the observer. By means of a special marking device, the outline of the heart, as the tube and screen are moved about, can be indicated at various points, either on the skin of the patient or on a sheet of paper. The examination of the patient can be made in the standing, sitting or lying position. The best forms of apparatus are the Groedel orthodiagraph designed for making tracings in the sitting position and Moritz's model for the horizontal position.

When plates are taken with the patient two meters from the source of light the error is about 0.5 cm. in any diameter, as the rays of light at this distance are nearly parallel. This is called teleradiography. It is the radiological method chiefly used in this country for measuring the size of the heart. Vaquez and Bordet insist that at this distance there is considerable deformity of the heart shadow. It is only at 2.5 meters they claim that the enlargement is so slight that it can be disregarded. For some reason orthodiagraphy has not received the recognition in America it deserves. In fact it has been but little used here by clinicians. Telefluoroscopy has some advantages over orthodiagraphy and is the method I have used for a number of years. A large fluoroscopic screen is placed in front of the subject who stands at a distance of two meters from the tube. Dr. L. B. Morrison of Boston has devised a convenient stand for holding this screen. A tracing is made on a large plate of glass which is fastened to the fluoroscope. The entire heart can be seen at one time. The small fluoroscope of the orthodiagraph gives a view of only a small portion of the border of the heart, and it has to be moved many times before the entire contour is traced. Telefluoroscopy is admirably adapted for the instruction of students. Its advantages over plates made at the same distance are, first, that the boundaries of the different portions of the heart can be more clearly made out in the pulsating organ itself and, second, the apex can usually be seen with the fluoroscope while in the

teleradiogram it is often lost to view below the level of the dome of the diaphragm.

Position of the Heart.—The heart is hung from the great vessels above and supported below by the diaphragm. The upper attachment is fixed, but the lower as it is formed by the diaphragm must move freely with it. As a result the position and also the shape of the heart varies with the position of the diaphragm. The position of the heart is dependent in part on the length of the suspensory apparatus formed by the great vessels, in part on the varying action of the weight of the heart in different positions of the body, but chiefly on the varying position of the diaphragm. The dome of the right half of the diaphragm stands higher than the left in both sexes and in every position. Both the right and left portion are as a rule higher in women than in men. The diaphragm is highest in the recumbent and lowest in the standing position. The apex is usually at the junction of the fifth interspace and the sixth rib when the subject is lying down; when sitting or standing it is an interspace or a rib lower. The apex as located by palpation is higher as a rule than its true position shown on the orthodiagram. Usually the difference amounts to an interspace.

If a heart were so small that it did not reach the diaphragm its long diameter would be vertical. Such a heart is never seen. Small hearts occur which are nearly vertical and rest on only a small portion of the diaphragm. A very large heart is in contact with the diaphragm over a large area. The apex and lower portion of the heart lie in the space between the dome of diaphragm and the anterior chest wall. The heart is forced into a horizontal position by the upward pressure of the diaphragm. A low diaphragm by lengthening the distance between the upper and lower points of attachment makes the heart assume a more vertical position. Lengthening of the aorta occurs in old age and this as well as a high position of the diaphragm by shortening the distance between the points of attachment causes the heart to become more or less horizontal.

The Normal Heart.—Examination is usually made in the dorsoventral position; that is with the patient's back toward the tube and the anterior portion of the chest in front of the observer. The heart, great vessels, spine and sternum form a middle dark shadow between the two light lung-fields. Above is the shadow formed by the tissues of the neck and below the diaphragm, liver and stomach. In the upper part of the stomach a large bright area due to air is often seen.

The right border of the silhouette formed by the heart and great vessels is in health made of two flattened arcs which join to form an obtuse angle at the level of the third rib. (Fig. 63a.) The border above this

point of junction is formed by the superior vena cava, the portion below by the right auricle. The contour of the right auricle is more convex

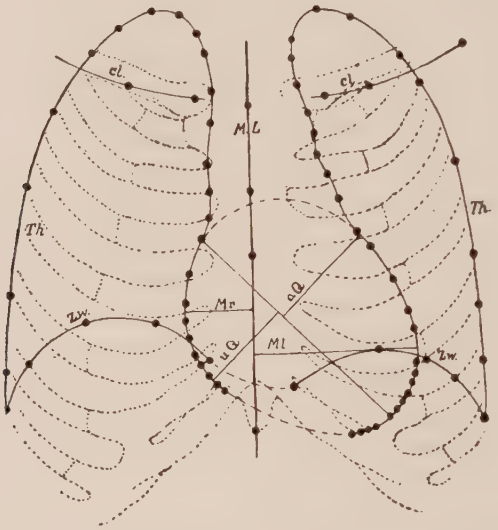


Fig. 63A.—Orthodiagram of heart with dorsoventral illumination. Ml median line; Mr distance of right border from median line; Ml distance of left border from median line; uQ + oQ = width (Breite) cl. clavicle; Th inner bony wall of thorax; Zw diaphragm (Seifert and Müller, "Taschenbuch der medizinsch-klinischen Diagnostik," J. F. Bergmann.)

than that of the superior vena cava and its lower part regularly forms an acute angle with the diaphragm—the cardiohepatic angle. Sometimes the right border of the heart is almost a straight line. The slight movement visible in the right auricle precedes the ventricular contraction. The superior vena cava shows no definite pulsation. There are three divisions of the left border. The upper of these at the level of the second rib is formed by the arch of the aorta. The second portion extends from the second to the third rib. It is usually a slightly concave sometimes a straight line. This middle portion of the left border is regularly formed by the pulmonary artery but the appendix of the left auricle may form its lower part. The third and lower portion made by the left ventricle is a distinct arc, a parabolic curve, which extends to the apex. The lower border of the heart, formed by the right ventricle, merges with the shadow cast by the diaphragm and liver. A line drawn from the junction of the superior vena cava and right auricle on the right border and the junction of the left auricle or pulmonary artery and left ventricle on the left border marks the base of the heart. Moritz draws between these two points a

curved line with its convexity upward. Above this line are the aorta, superior vena cava, pulmonary artery, and left auricle, below the line are the two ventricles and the right auricle.

Standard Measurements. A series of standard measurements are made from the silhouette of the heart and great vessels. The median line is drawn on the tracing and the position of the clavicles and the nipples is indicated. A line connects the median line with the most distant point of the right border. This is called Mr. The most distant point on the left border is connected with the median line by a straight line. This is termed Ml. The sum of the two gives the transverse diameter. Another important measurement is the length of the heart. This is the distance from the junction of the right auricle and superior vena cava above to the junction of the left and lower borders below (the apex). It is less than the true length of the heart as the ventricles extend forward as well as downward. This causes the shadow to be slightly foreshortened. In the orthodiagram and telefluorogram the position of the apex usually can be made out readily when the diaphragm descends. If not seen on quiet breathing a deep inspiration generally brings it into view. In teleradiograms the apex as already stated often lies in the dome of the diaphragm and hence cannot be located. The tracing of the cardiac outline should be made in the position of diastole during ordinary breathing. It is a mistake to outline the heart in full inspiration, as in this position the heart becomes more vertical, the divisions of its borders less distinct, and the length increased at the expense of the transverse diameter. In the tables, the length of the heart is indicated by the letter L. Another measurement of distinct value in certain cases is the greatest distance of the right border and the left border from the longitudinal diameter. This is termed Br, the Breite (width) of the heart, by Moritz. It should not be confused with the transverse diameter which is the width of the heart in relation to the chest but not the true width of the heart itself.

Size of the Normal Heart.—The heart shadow is largest with the body in the supine position, smaller in the sitting posture and smallest when standing. As teleradiograms and telefluorograms are made with the subjects standing, Otten's tables of normal measurements are more serviceable than those of Dietlen made with persons recumbent and those of Groedel which were taken in the sitting position.

TABLE I

Size of the Heart (Otten)

STANDING

MEAN VALUES FOR ADULT MEN

Height	Mr	Ml	Tr	L	Br
155–164 cm.	4.1 cm.	8.3 cm.	12.4 cm.	13.7 cm.	9.4 cm.
165–174 "	4.0 "	8.5 "	12.5 "	13.8 "	9.6 "
175–184 "	4.2 "	8.5 "	12.7 "	14.4 "	9.8 c

MEAN VALUES FOR ADULT WOMEN

Height	Mr	Ml	Tr	L	Br
145–154 cm.	3.6 cm.	7.5 cm.	11.1 cm.	12.7 cm.	8.6 cm.
155–164 "	3.6 "	7.6 "	11.2 "	12.9 "	8.8 "
165–174 "	3.7 "	7.8 "	11.5 "	13.0 "	8.9 "

The size of the heart as shown in these and other tables varies with the height of the individual and the sex. It is smaller for women than for men of the same height. The size varies normally with the age and the circumference of the chest as well as with the height and weight of subjects. This is well shown in the average figures obtained by Hammer and Rieder as given in Seifert and Müller's work.[12]

TABLE II

Size of the Heart (Hammer and Rieder)

	Mr	Ml	Tr
ACCORDING TO HEIGHT			
154–160 cm.	4.3 cm.	8.5 cm.	12.8 cm.
160–170 "	4.8 "	8.7 "	13.5 "
170–180 "	4.8 "	8.7 "	13.5 "
180–184 "	4.4 "	9.6 "	14.0 "
ACCORDING TO WEIGHT			
50–60 kgm.	4.3 cm.	8.3 cm.	12.6 cm.
60–70 "	4.6 "	8.5 "	13.1 "
70–80 "	5.2 "	8.9 "	14.1 "
ACCORDING TO AGE			
20–30 years	4.7 cm.	8.7 cm.	13.4 cm.
30–40 "	4.9 "	8.8 "	13.7 "
40–50 "	5.7 "	9.6 "	15.3 "

[12] Seifert and Müller, "Taschenbuch der medizinisch-klinischen Diagnostik," 1921, 66.

According to the Circumference of the Chest			
80–85 cm.	4.6 cm.	8.3 cm.	12.9 cm.
85–90 "	4.5 "	8.6 "	13.1 "
90–95 "	4.9 "	9.0 "	13.9 "
95–100 "	5.0 "	9.0 "	14.0 "

There is a great individual variation in the measurements of subjects of the same height and weight so that slight enlargement of the heart unfortunately cannot be determined by measurements of the orthodiagram. In some cases of definite hypertrophy and dilatation occurring in mitral stenosis and aortic insufficiency of mild degree the size of the heart is less than in some normal individuals of the same height. This is clearly brought out by the maximum and minimum figures obtained by Dietlen in normal persons which are collected in the following table.

TABLE III

Maximum and Minimum Measurements of the Normal Heart (Dietlen)

Height	Mr	Ml	Tr	L	Br
Men (20 Years and Over)					
145–154 cm.	3.1–4.4 cm.	8.2– 8.8 cm.	11.9–12.6 cm.	12.1–14.1 cm.	8.5–10.5 cm.
155–164 "	3.3–5.9 "	7.4–10.4 "	11.0–14.5 "	12.3–15.3 "	9.1–11.0 "
165–174 "	3.0–5.7 "	6.8– 9.7 "	11.3–15.3 "	12.5–15.9 "	9.2–11.7 "
175–187 "	3.5–5.8 "	8.1–11.0 "	13.1–15.0 "	13.4–16.2 "	10.0–11.4 "
Women (17 Years and Over)					
145–154 cm.	2.4–4.0 "	7.2– 9.2 "	10.3–12.8 "	12.1–13.3 "	8.6–10.2 "
155–164 "	2.6–5.2 "	6.8–10.3 "	10.9–13.7 "	11.7–15.0 "	8.6–10.6 "
165–174 "	3.2–4.5 "	6.8– 9.7 "	11.3–12.9 "	12.8–14.0 "	9.3–10.6 "

Changes in the form of the cardiac silhouette are often of greater aid in diagnosis than the increase in size. In some cardiac diseases the form of the heart shadow is so characteristic that a conclusion can be made from it with considerable confidence regarding the site of the disease and the presence of a dilatation.

The measurements vary somewhat with the position of the heart. If the diaphragm is very high, that is if it reaches to the fourth rib or the third interspace on the right side, the heart becomes more horizontal. As a result the transverse diameter (Mr and Ml) increases and the length (L) decreases. The long diameter forms a narrow angle with the horizontal. If the chest is long and the diaphragm low, the heart becomes vertical. The transverse diameter is less than the normal mean figure and the angle between the long diameter and the horizontal plane becomes wider.

There are three types of the normal: the oblique, the vertical and the horizontal (Dietlen). These three types have as a definite fundamental form an oval placed more or less obliquely to the median line of the thorax, with a short rounded pole above and to the right and a longer pointed pole below and to the left.

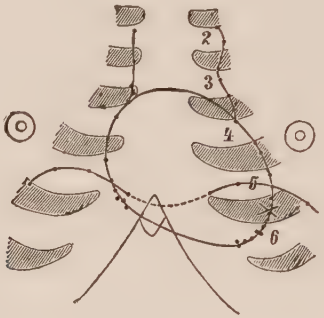


Fig. 64.—The oblique heart. (Dietlen, Deutsches Arch. f. klin. Med. Leipz.)

The *oblique* heart (Fig. 64) is found in the great majority of men with normal thoraces. The oval of the heart shadow is rather long, the apex narrow and lies almost always below the left dome of the diaphragm. The angle the long diameter forms with the horizontal measures 35° to 38°.

The *vertical* type (Fig. 65) occurs chiefly in young people especially in those with a narrow thorax; in pronounced form it appears as the pendu-

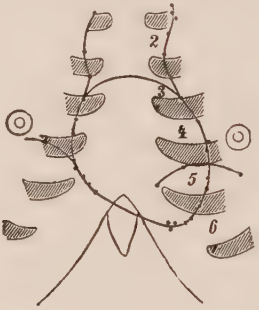


Fig. 65.—Vertical heart. (Dietlen, Deutsches Arch. f. klin. Med. Leipz.)

lous or dropped heart. The apex is lower in the chest and near the median line, lying without exception beneath the left dome of the diaphragm. The inclination angle of the longitudinal diameter to the horizontal is 39°–43°. The heart appears narrower and longer than the obliquely placed heart.

The *horizontal* type (Fig. 66) is found regularly in thickset persons of medium size with a broad short thorax and is common in old people. The inclination angle varies between 25° and 35°. The apex is more rounded and lies often above the dome of the diaphragm. The heart appears broader and shorter than in other types. Usually the heart in women is of the horizontal type.

The frequency of the occurrence of the different types of heart varies considerably with the position of the body selected for examination. The oblique is more frequent in the sitting position, the horizontal in the lying,

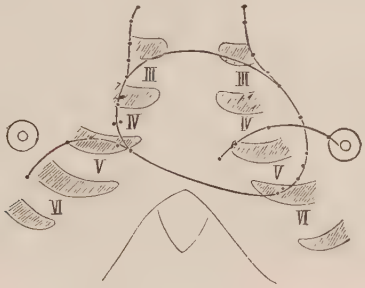


FIG. 66.—Horizontal heart. (Dietlen, Deutsches Arch. f. klin. Med. Leipz.)

and the vertical when the subject is standing. In a series of 100 normal persons in the standing position, the heart was of the vertical type in 62 per cent; of the horizontal in 21, and of the oblique in 17 per cent. (Otten.)

Instead of comparing the measurements of the heart in various diameters with standard tables the attempt has been made to recognize enlargements by determining the ratio of the greatest horizontal diameter of the heart to the internal diameter of the chest. Normally in men this is 1 : 1.92. The broad statement is generally true that a heart that measures transversely 50 per cent or more of the internal diameter of the chest is dilated. The value of this method of determining cardiac enlargement is lessened by its failure to take into account the variations in the position of the heart. A heart of normal size of the horizontal type would have a greater transverse diameter and hence represent a larger percentage of the internal diameter of the chest than a larger heart that was of the vertical type.

The Dropped Heart.—The heart is long, narrow and pendulous in many patients taken in the standing position if the plate or tracing is made when the breath is held at the end of a full inspiration. Ignorance of this fact has frequently led to the diagnosis of a dropped or ptotic heart when the shape of the heart in quiet breathing was perfectly normal.

Pendulous hearts do occur that are regarded as pathological. Their form is an exaggeration of the vertical heart described above. (Fig. 67.) They occur in subjects with long narrow chests. They are more

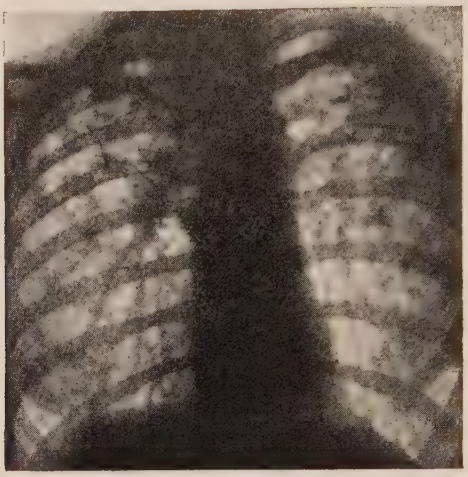


Fig. 67.—Dropped heart. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

frequent in young than in older persons and are usually associated with ptosis of the abdominal organs. For a time it was thought that the re-

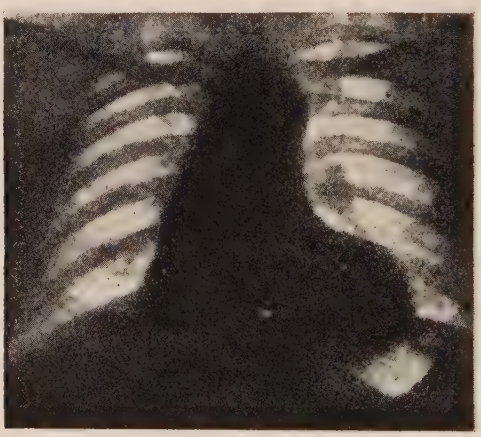


Fig. 68.—Aortic insufficiency. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

serve cardiac power of subjects with ptotic hearts was lessened, but observations made during the world war showed this was not necessarily true. (Brugsch and Schittenhelm.) Certain cases of dyspnea on exer-

tion are apparently due to ptosis of the heart as the symptom disappeared when the position of the diaphragm was elevated by an abdominal binder (Wenckebach). Very few of these cases have been observed and reported. It is undoubtedly true that the dropped heart as a cause of symptoms has very little significance; much less than has been attached to it by some writers.

Aortic Insufficiency.—The heart is horizontal and extends far to the left owing to dilatation of the left ventricle. The alteration in form

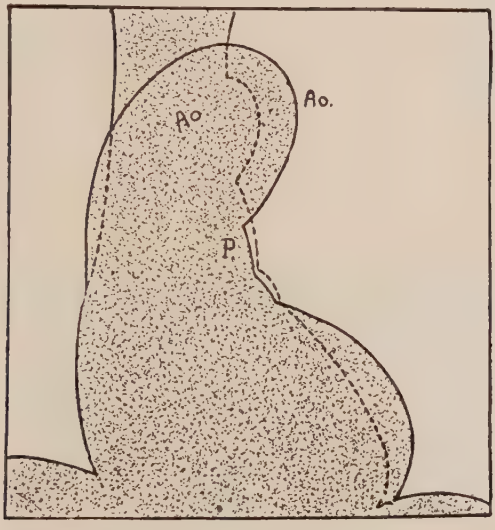


Fig. 69.—Type of the shadow in aortic insufficiency. The dotted line is the outline of the normal silhouette. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

makes the heart assume the shape of a shoe. (Fig. 68.) The apex becomes rounded. Increase in the length of the left median distance (M1) soon becomes noticeable. The first portion of the aorta widens and forms the right border of the great vessels by replacing the superior vena cava. The aorta often gives a more definite convexity to the upper part of the right border. An outward movement of this arc systolic in time (Fig. 69) occurs when it is formed by the aorta; normally as already stated it is almost motionless.

Dilatation of the Left Ventricle.—This occurs frequently in the absence of aortic insufficiency in chronic Bright's disease, essential hypertonia, and cardiosclerosis. The shape of the cardiac shadow may resemble somewhat that of aortic insufficiency for which it is sometimes

mistaken. (Fig. 70.) Dilatation of the right ventricle is often associated with that of the left ventricle and this combination increases markedly the transverse diameter of the heart.

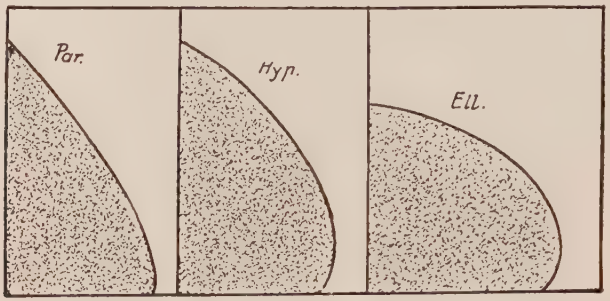


FIG. 70.—Diagrammatic sketch of the deformities of the contour of the left ventricle. Par. Normal parabolic form; Hyp. Hyperbolic curve when dilatation predominates; Ell. Elliptical form when hypertrophy predominates. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

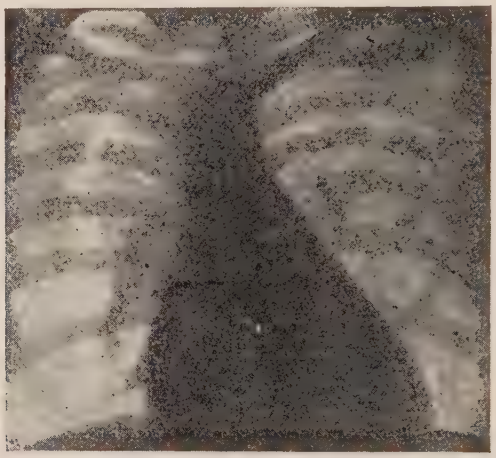


FIG. 71.—Mitral heart. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

Mitral Stenosis.—In early mitral disease, the diagnosis is suggested by the altered form of the heart shadow rather than by an increase in the measurements. (Fig. 71.) The middle arc of the left border may become prominent due to dilatation of the left auricle without any increase of the diameter Br or the other standard orthodiagraphic measurements. (Fig. 72.) In more severe cases especially when cardiac insufficiency has developed the Mr is increased and the right border of the heart becomes more convex. Sometimes this border, especially in its lower part,

is formed by the right ventricle when this chamber is dilated. If so, a wider movement of the border occurs and it is systolic in time. If there

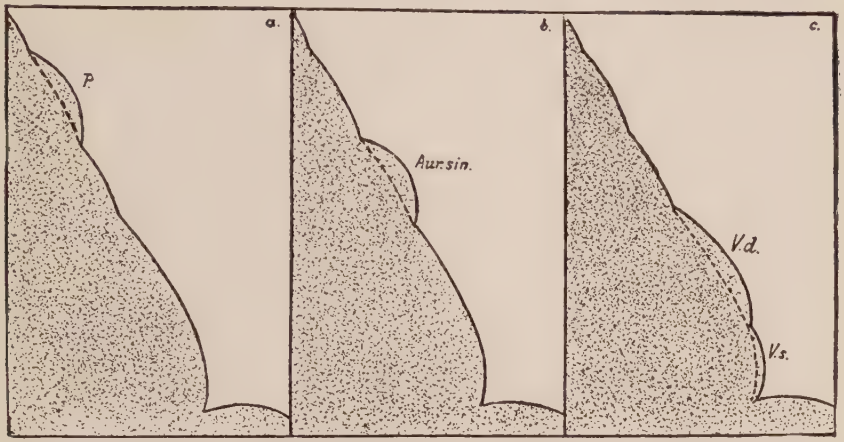


FIG. 72.—Prominence on the left border of the heart silhouette in mitral disease. (a) prominence of the dilated pulmonary artery; (b) prominence of the dilated left auricular appendage; (c) prominence of the dilated right ventricle. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

is little or no mitral regurgitation combined with the stenosis the left ventricle will remain small. In advanced mitral stenosis and insufficiency with cardiac weakness the heart becomes nearly spherical in shape, owing

FIG. 73.—Mitral heart with prominence of the pulmonary artery. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

to general enlargement of all the four cavities, and the silhouette in the dorsoventral position almost circular. (Fig. 73.)

Cardiosclerosis.—The form of the heart is not characteristic. It is usually horizontal, and there is generally dilatation of the left ventricle and often of the right. As a result the heart is increased in both the longitudinal and transverse diameters but in different proportion in different cases. The aorta is often wide and the contour of the upper portion of the right border may be more convex and the first arc on the left side more prominent.

Congenital Heart Disease.—The most frequent form is the *patent ductus arteriosus Botalli.* Characteristic of this disease is the bulging of the second arc on the left border due to enlargement of the pulmonary artery. The dilatation of the right ventricle pushes the left lower arc formed by the left ventricle upward and outward. In *congenital pulmonary stenosis* there is enlargement of the right ventricle. There may be a distinct bulging of the right border of the heart after the onset of cardiac failure.

Pericarditis.—The greatest enlargement of the cardiac shadow is seen in *pericardial effusion.* The different arcs can no longer be distinguished and when the effusion is massive the shape of the silhouette is almost circular, with the great vessels forming a short thick pedicle above. In some cases the figure has been compared to an onion which it somewhat resembles. The left and right borders spread out suddenly from their junction with the great vessels. The left border may run in almost a horizontal direction to the thoracic wall if the effusion be very large. The transverse diameter of the shadow is greatly increased especially in comparison with the longitudinal diameter (Vaquez and Bordet). If the effusion is smaller in amount the heart shadow may closely resemble that seen in general dilatation of the heart.

The pulsation of the heart in pericardial effusion when viewed with the fluoroscope is much decreased or even absent. It is not always possible to distinguish between the silhouette of pericardial effusion and that of marked dilatation of the heart. In the latter condition, however, the visible pulsations may be slight but they never disappear completely.

Pleuropericardial bands and adhesions *in adhesive pericarditis* are sometimes seen to form definite shadows. One or both of the cardiophrenic angles may be filled with ribbon-like opacities, and the contour of the heart shadow marked by projections or indentations. The cardiac pulsation viewed with the fluoroscope may be lessened because of broad adhesions covering the heart borders and the movement of the diaphragm be diminished (Lehmann and Schmoll).

Syphilitic Aortitis.—If this disease is suspected, the examination should never be regarded as complete without a careful radiological ex-

amination of the aorta. In this study radiograms or orthodiagrams should be made in the first oblique diameter as well as in the dorsoventral position. I have found telefluoroscopy admirably adapted to the study of the aorta. Using a large screen, the aorta can readily be examined in the oblique diameters and its outline traced. The first oblique diameter (Holzknecht) is also called the right anterior oblique position (Vaquez and Bordet). It should always be employed in examining the ascending aorta and the arch of the aorta as it usually permits an earlier recognition of localized dilatation of the aorta that can be obtained by other means. The subject is turned to an angle of from 35° to 50° so that the right shoulder is brought near the screen but the left is moved away from the screen and the observer. The rays enter the left posterior thoracic wall

FIG. 74.—View of heart and aorta in first oblique diameter (Holzknecht) right anterior oblique diameter (Vaquez and Bordet) *R*, right lung-field; *M*, posterior mediastinum, *A*, aorta; *H*, heart; *L*, left lung-field. (Morawitz, Klinische Diagnostik innerer Krankheiten.)

and come out through the right thoracic region. The angle of obliquity of the body can be measured by the easily constructed angle indicator or goniometer described by Vaquez and Bordet.[13] A diagrammatic sketch of the view obtained in the first oblique diameter is shown in Fig. 74. The broad curved shadow extending from the top to the bottom of the thorax is the spine. Between this and the heart shadow are the posterior mediastinal tissues and a portion of the right lung. Above the heart is the aorta which in the orthodiagram is a tongue-shaped shadow about the thickness and size of two thumbs.

The posterior border of the heart at the angle of 50° shows both the left and right auricles, at 45° and less only the right auricle, with the right ventricle below it. In aortitis the aorta becomes wider and the

[13] Vaquez and Bordet, "The Heart and the Aorta," translated from the second French edition by Honeij and Macy, New Haven, 1920, 39.

upper end of the shadow in the oblique diameter often presents a bulbous swelling. The aortic shadow is usually much darker than normal. The aorta becomes increased in length as well as in width. The changes in

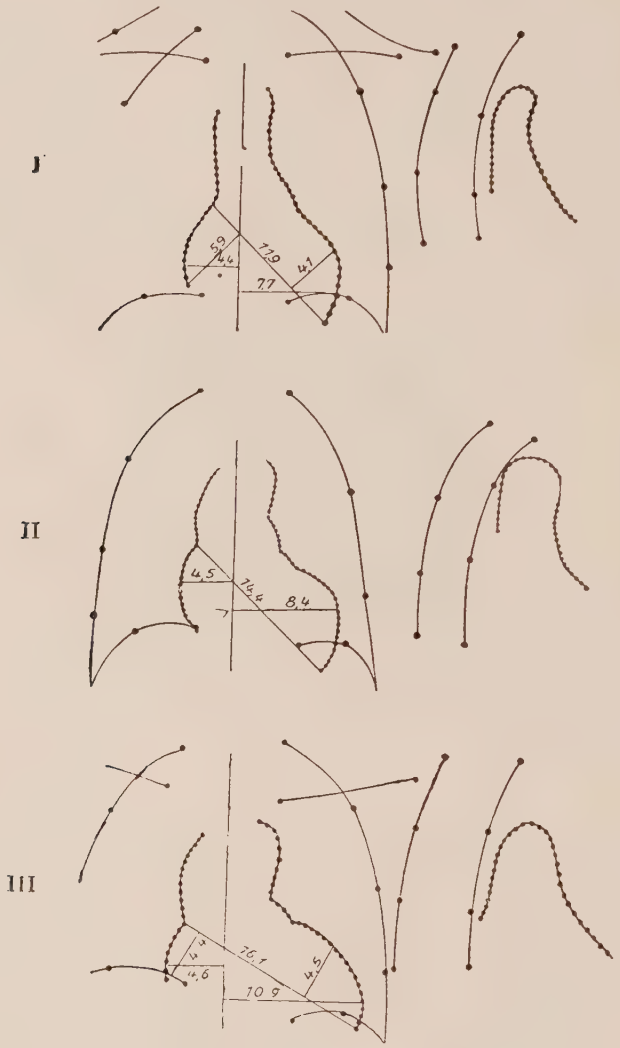


FIG. 75.—Orthodiagrams of the heart and aorta in syphilitic aortitis, with transillumination made in both the dorsoventral and the first oblique diameters. I, normal length and width, but increased curving of the aorta; II, beginning elongation and dilatation of the aorta; III, marked elongation and dilatation of the aorta, displacement downward of the right heart. (Romberg, "Krankheiten des Herzens und der Blutgefässe," Ferdinand Enke.)

the shape of the aorta in syphilitic aortitis are well shown in the orthodiagrams reproduced in Fig. 75.

Aneurism.—In aneurism of the thoracic aorta, the dilatation produces a marked bulging of the contour of the shadow. Orthodiagrams or tele-

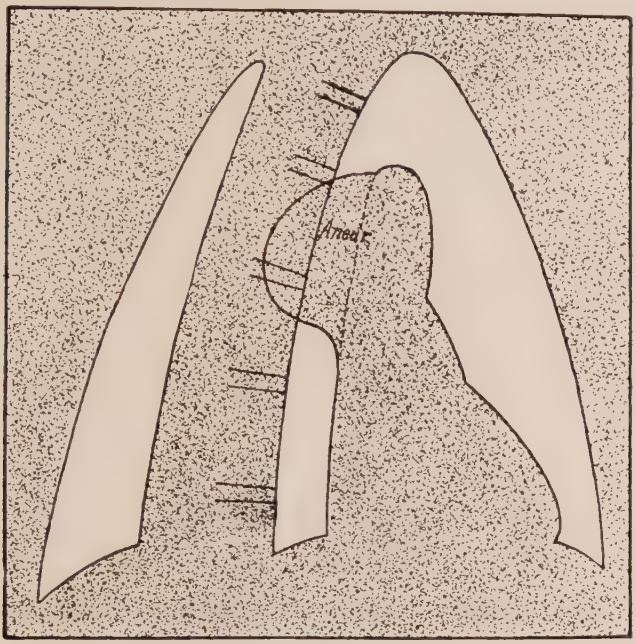


FIG. 76.—Aortic aneurism seen in first oblique diameter. (Schwartz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

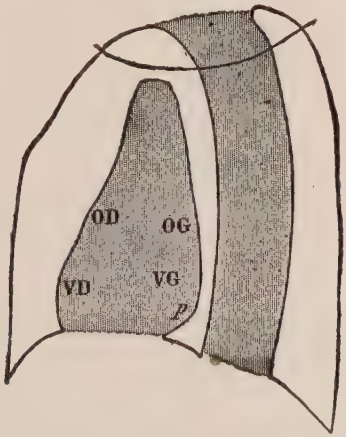


FIG. 77.—Orthodiagram taken in the second oblique diameter (left anterior oblique position of 50 degrees). OD, right auricle; OG, left auricle; VD, right ventricle; VG, left ventricle; p, apex of the heart. (Vaquez and Bordet, "The Heart and the Aorta," Yale University Press.)

radiograms should be made with dorsoventral illumination, and also in the first oblique diameter. (Fig. 76.) The second oblique diameter

(Holzknecht) called also the left anterior oblique position (Vaquez and Bordet) is occasionally useful in detecting the existence and location of aneurisms. The patient's left shoulder is near the screen and the illu-

Fig. 78.—Aneurism of the ascending portion of the aorta. (Vaquez and Bordet, "The Heart and the Aorta," Yale University Press.)

mination extends through the chest from the right back to the left front. See Fig. 77. A lateral orthodiagram is at times of value in the diagnosis of aneurism. The right or left shoulder is in contact with the screen so that the illumination passes from one lateral region of the chest to the

Fig. 79.—Same case, in the first oblique diameter (right anterior oblique position). (Vaquez and Bordet, "The Heart and the Aorta," Yale University Press.)

other. The biscapular axis forms an angle of 90 degrees. By combination of the pictures made in the different diameters definite knowledge can be obtained of the situation and the form of the aneurism. The most common location is in the ascending aorta. An aneurism here usually causes a projection of the first portion of the right border of the cardiovascular silhouette. Orthodiagrams of aortic aneurisms are shown in Fig. 78 and Fig. 79.

Aortic Sclerosis.—Examinations should be made in the first and second oblique diameters as well as in the dorsoventral. Sclerotic processes convert the narrow cylindrical tube of the aorta into a more or less bul-

FIG. 80.—Arteriosclerosis. Heart and aorta viewed in second oblique diameter (left anterior oblique position). The descending aorta is visible. (Gerhartz, "Leitfaden der Röntgenologie," Urban and Schwarzenberg.)

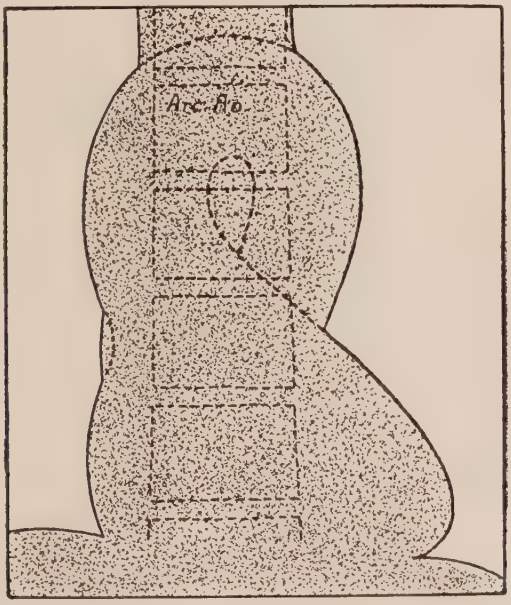


FIG. 81.—Diagram of the dilated aorta. (Schwarz, "Handb. der Herz-u. Gefässerkrankungen," Franz Deuticke.)

bous swelling. The entire thoracic aorta is often abnormally wide in an early stage of the disease. (Fig. 81.) The shadow of aortic sclerosis differs from that of luetic aortitis in that the increase in diameter is less

localized to a single portion of the aorta in the former but the distinction between the two conditions cannot be made by radiograms alone.

SPIROMETRY

By vital capacity of the lungs is understood the greatest voluntary expiration following the deepest inspiration (Hutchinson). It is measured by a simple instrument called a spirometer, which usually consists of an inverted cylinder submerged in an outer chamber containing water. The expired air is conducted by a flexible pipe and central tube into the inner chamber, which rises in the water. An index marks the cubic centimeters expired after a forced inspiration. (Fig. 82.) The vital capac-

Fig. 82.—Position of the body in filling the chest before breathing into the spirometer to measure the vital capacity of the lungs. (Hutchinson, Med.-Chir. Tr., Lond.)

ity varies normally with height, weight, age and sex. It attains its maximum between the age of 20 and 30 and then slowly decreases. It is considerably lower in women than in men of the same height and age.

West [14] found a more constant relationship existed between the vital capacity and the body surface area than between the vital capacity and the height, weight or chest volume. The standard he recommends is determined by dividing the vital capacity by the surface area of the body. The latter is calculated by Du Bois, formula. By means of the graphic

[14] *Arch. Int. Med., Chicago,* 1920, XXV, 306.

chart published by Du Bois and Du Bois,[15] the surface area is easily determined when the height and weight are known. (Fig. 83.) According to West, the normal vital capacity for men is 2.5 liters per square meter of body surface, for women 2 liters.

In Table IV, the influence of age on the vital capacity is shown.

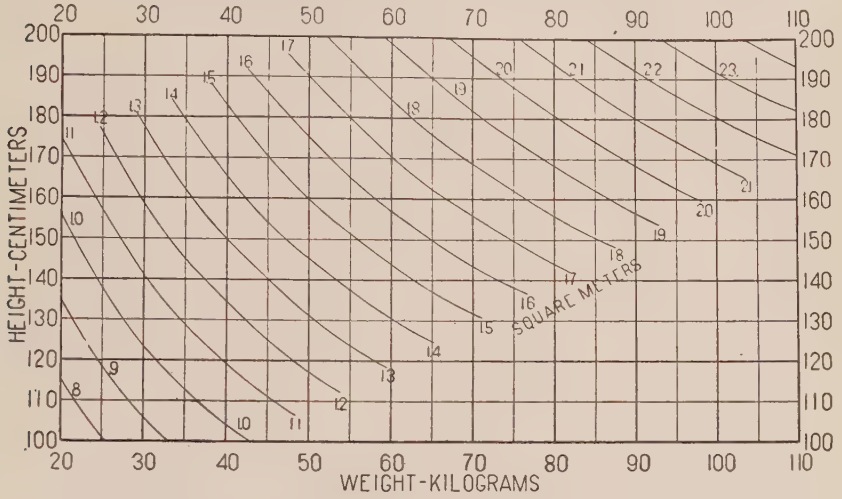


Fig. 83.—Chart for determining surface area of man in square meters from weight in kilograms (Wt.) and height in centimeters (Ht) according to the formula: Area (Sq. Cm.) = Wt. 0.425 X Ht. 0.725 X 71.84. (Du Bois and Du Bois, Arch. Int. Med., Chicago.)

TABLE IV

Age	No. of persons	Average height in cm.	Average Weight in kgm.	Average Body surface area in sq. m.	Average vital capacity in cc.	Average per cent of West's standard
10 to 20	9	157	47.0	1.47	3125	82
20 to 30	14	173	58.6	1.72	4500	105
30 to 40	23	166	67.1	1.75	3950	90
40 to 50	20	171	69.0	1.81	3775	83
50 to 60	20	170	73.9	1.85	3825	83
60 to 70	11	169	69.8	1.80	3300	73
70 to 80	3	172	69.9	1.82	2525	56

Fig. 84.—Personal observations on the influence of age on vital capacity in 100 normal men. (Pratt, Am. J. M. Sc., Phila., CLXIV.)

The vital capacity is low in cardiac insufficiency because of the inability to increase the depth of breathing in the normal manner (Peabody). This decrease is found even in cases which present no clinical evidence

[15] *Ibid.*, 1916, XVII, 863.

of chronic passive congestion of the lungs. When patients improve under treatment there is a rapid and marked increase in the vital capacity.

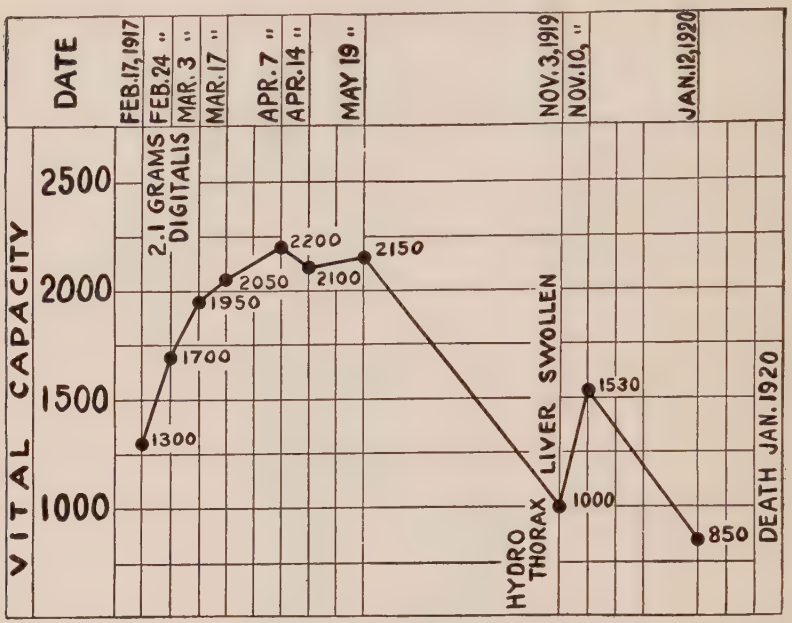


Fig. 85.—Mitral stenosis with auricular fibrillation. Improvement from continued administration of digitalis leaf without bed rest. Record of vital capacity for period of nearly three years. (Pratt. Am. J. M. Sc., Phila.)

An increase of cardiac weakness is accompanied by a further fall in vital capacity. Personally I have made determinations of the vital capacity in a large number of patients with heart disease and have found it to be a good index of the amount of reserve power possessed by the heart. The vital capacity is reduced in various lung diseases especially in bronchial asthma during the attacks. It is, of course, an indirect measure of the cardiac power. But for years, physicians have recognized that the condition of a patient's wind is the best guide to the state of the heart. This the spirometer permits us to measure with some precision, and without subjecting bed patients to injury that might result from the ordinary exercise tests. Relatively little importance can be placed on single isolated observations. In women, especially, the vital capacity may be low even when the heart and lungs are normal.

In measuring the increase or decrease of cardiac insufficiency in a case from day to day, or week to week, the vital capacity determinations are of great value. A vital capacity chart such as shown in Fig. 85, gives a far better idea of the condition of the heart and circulation during the period of observation than any other form of graphic record.

CHAPTER IV

CARDIAC IRREGULARITIES

The impulse to contraction normally develops in the sino-auricular node situated at the junction of the right auricle and superior vena cava (see page 245). This function of the node is influenced by the vagus nerve which tends by its action to decrease the rate of impulse formation and by the accelerator nerve which stimulates the node to form impulses more rapidly. More is known of the action of the vagus than of the accelerator. The vagus by restraining stimulus formation in the sinus node tends to hold the heart in leash. As the two auricles contract at the same time and the two ventricles contract also simultaneously, but later than the auricles, we can consider the heart physiologically as if composed of one auricle and one ventricle. Under abnormal conditions the impulse to contraction may form in the auricular muscle, in the auriculoventricular node (page 246) or in the ventricular muscle.

The arrhythmias can be classified according to the place of origin of the impulse. There are only the four areas already mentioned in which the impulse can arise, and irregularities due to impulse formation in each of them have been observed clinically.

It is convenient clinically to divide the irregularities of the heart under the following eight types:

(1) Sinus arrhythmia.
(2) Extrasystoles or premature contractions.
(3) Paroxysmal tachycardia.
(4) Auricular flutter.
(5) Auricular fibrillation.
(6) Disturbances of conduction (heart-block).
(7) Pulsus alternans.
(8) Pulsus paradoxus.

SINUS ARRHYTHMIA

In this form of cardiac irregularity there generally is a gradual waxing and waning of the pulse-rate. It is the result of variation of the vagus inhibition of impulse formation in the sinus node. It occurs in typical

form in healthy children and then is associated with the act of respiration. It also is present in young adults during deep breathing. The pulse quickens during inspiration and slows during expiration. It is the slow pulse during the expiratory period that usually attracts attention, and failure to recognize the nature of the irregularity has often led to the mistaken diagnosis of heart disease in young children. Most of the arrhythmias occurring during the first decade are of this type. It is also common at the age of puberty and has been termed the juvenile arrhythmia by Mackenzie. When irregularity is marked during quiet breathing it indicates increased irritability of the vagus.

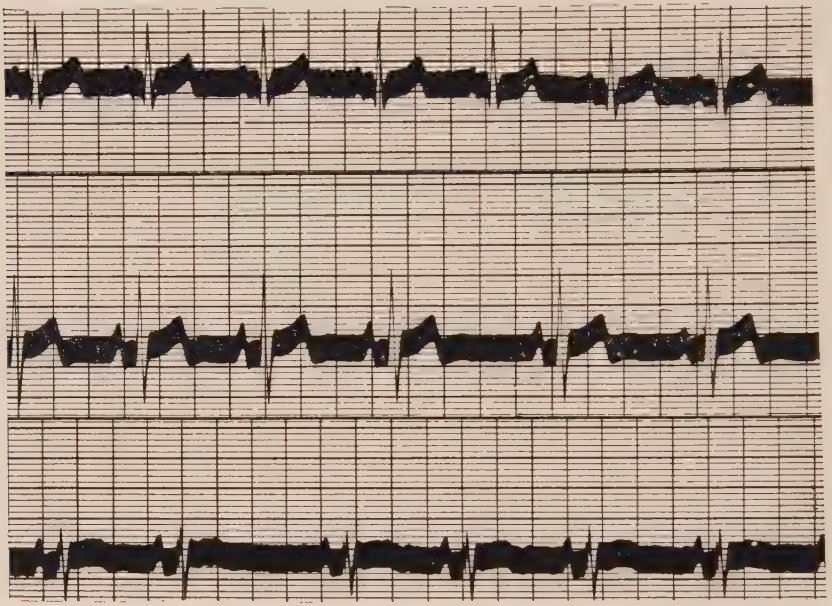


Fig. 86.—Sinus arrhythmia. The auricular complexes are normal but the intervals between them vary in length. (Dr. Paul D. White.)

Sinus irregularities also occur that bear no relation to respiration. These are usually associated with a reduction of the pulse-rate. In one form the phasic variation of the pulse-rate with its slowing and subsequent acceleration of the heart occupies a period of ten or more seconds. (Lewis.) In Cheyne-Stokes breathing there is usually bradycardia with hyperpnea and tachycardia with apnea.

If there is a definite relation of the irregularity to respiration no additional evidence is really necessary to decide that it is a sinus arrhythmia. It has been confused with pulsus paradoxus in which the size of the pulse waves varies with respiration, but in pulsus paradoxus although the pulse

waves are smaller during inspiration the separate beats may be evenly spaced. A recent writer maintained that pulsus paradoxus was only a form of respiratory arrhythmia. The tracings published to support his views were examples of sinus arrhythmia with a quickening of the rate during inspiration. In true pulsus paradoxus as I know from personal observation, see Fig. 109, page 442, the rhythm may be regular and only the size of the waves may vary with the phase of respiration.

If in sinus irregularity there is no relation to respiration apparent it can be brought out on deep slow breathing. The radial pulse waves are of good size and of equal height as shown in radial tracings. When the pulse-rate is considerably accelerated sinus irregularities disappear. This can be produced by exercise, amyl nitrite or atropine. Likewise fever restores the regular rhythm. A sinus irregularity is not a manifestation of disease. Its existence is said to indicate instead the integrity of the heart muscle (Mackenzie, Wenckebach), but this is not strictly true. It rather means an intact or fairly normal nervous mechanism. It does not indicate the condition of the muscle. Since, however, it is more common in pronounced degree in young people and a healthy heart muscle is more common in young people than in old, this confused idea has arisen. It may even be found in pulsus alternans. (White.) Its chief importance consists in the possibility of confusing it with an irregularity of serious significance.

EXTRASYSTOLES

The auricles, the auriculoventricular node and the ventricles have the latent property of impulse formation. Stimuli to contraction under abnormal conditions may arise in any of these three structures, if any point in them becomes more excitable than the sino-auricular node. An extrasystole may be defined as a premature contraction of the heart that is independent of the normal rhythm and arising in response to an impulse originating in some part of the heart other than the sinus node. The normal contraction is due to a single impulse that is formed in the sino-auricular node and requires a relatively long time for its development when the heart is beating at a normal rate according to Lewis. The extrasystole requires little or no time for its preparation and to this quality its prematurity is due. This is denied by other authorities.

Neither the auricle nor the ventricle respond to stimuli during the period of contraction. This non-excitable condition is called the refractory state. Stimuli applied after early diastole to either auricle or ventricle produce a contraction.

Auricular Extrasystoles.—After a premature contraction of the

auricle the normal auricular rhythm is disturbed. The extra stimulus is apparently conducted back to the sinus node and destroys the partly prepared impulse for the next contraction. (Cushny, Wenckebach.) The pacemaker in the node then begins to prepare a new normal impulse and the time required is the interval between two normal auricular contractions.

The premature contraction of the auricle is followed by a premature contraction of the ventricle. (Fig. 87.)

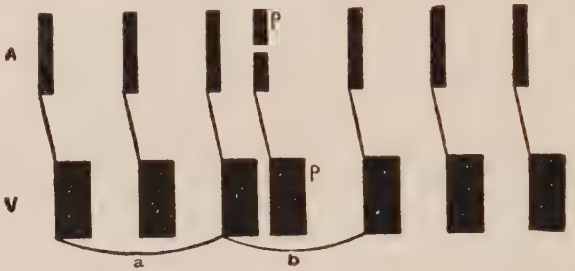


FIG. 87.—A premature auricular contraction is represented diagrammatically. The auricular rhythm is disturbed by the abnormal auricular beat (*p*); the disturbance in the ventricular rhythm is parallel, for each auricular systole yields a ventricular response. The rhythm of the whole heart is dislocated, the period *a* is longer than the period *b*. (Lewis, "Clinical Disorders of the Heart Beat," Shaw and Sons.)

The ventricular beat is followed by a pause, but the interval occupied by the normal beat plus the following extrasystole (b) is not equal to

FIG. 88.—Shows two premature or extrasystoles of auricular origin (x). The waves *c'* in the jugular tracing occur at the same time as the small premature beats (x) in the radial tracing, and are therefore due to the carotid. These are preceded by premature waves *a'* due to the auricle. The interval *a'–c'* (space A3) is greater than the average *a–c* interval (A2), and is much greater than the following *a–c* interval (A4). (Mackenzie, "Diseases of the Heart," Oxford University Press.)

full cycles of the normal rhythm (a). If the auricular extrasystole originates late in the diastole of the auricle the interval is almost equal to two normal beats; if the extrasystole arises early in diastole the interval is always distinctly less. (Hirschfelder and Eyster.)

Ventricular Extrasystoles.—If an extrasystole arises in an irritable focus in the ventricle it usually happens that the next normal impulse to contraction that reaches the ventricle from the auricle arrives while the ventricular muscle is in a refractory state and hence fails to produce a

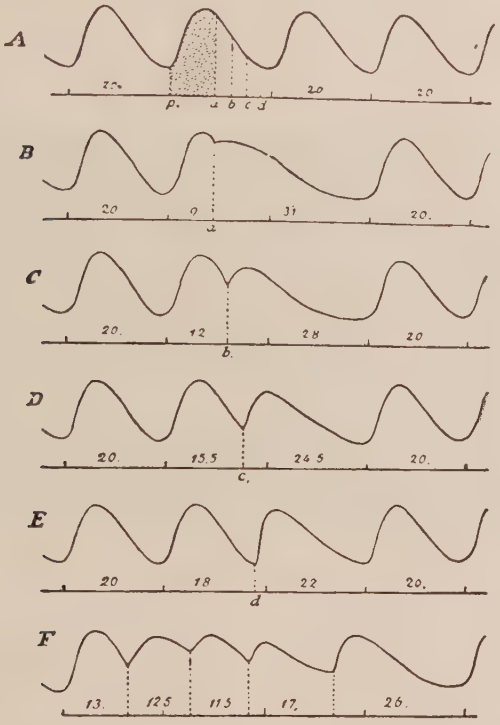


FIG. 89.—Extrasystoles. A represents a series of normal contractions of the ventricle. The refractory period begins (p) just before the onset of systole and ends at *a*. At this point *a* the heart muscle again becomes excitable for strong electrical stimuli; at *b* a less strong stimulus suffices to produce systole; at *c* a still weaker one and at *d* just before the succeeding systole, the excitability of the muscle is entirely restored and again responds to a weak stimulus. The effect of stimulation at *a b c* and *d* is represented diagrammatically in B, C, D and E. At *a* the contractility has been so little restored that the extrasystole is very small. Stimulation at *b, c, d* produces progressively larger extrasystoles (B, C, D and E). In the curves B to E, the compensatory pause is also represented. In F is shown a series of extrasystoles followed by a spontaneous systole which occurs at the moment which it would have appeared if the heart rhythm had not been disturbed. (Wenckebach, "Die Arhythmie," W. Engelmann.)

contraction. (Fig. 89.) The period of the disturbance is equal to two full cycles of the normal rhythm. The diastole following the extrasystole is unusually long, as the ventricle must wait until the arrival of the next

normal stimulus from the auricle. The increase in length of the diastole compensates for the short diastole that preceded it and is called the compensatory pause. After this the regular rhythm may be restored. The auricular rhythm is not usually disturbed by the ventricular extrasystoles but sometimes it is by retrograde conduction. Occasionally the ventricular extrasystole is interpolated between two normally timed ventricular contractions if it occurs early in diastole and the heart-rate is slow.

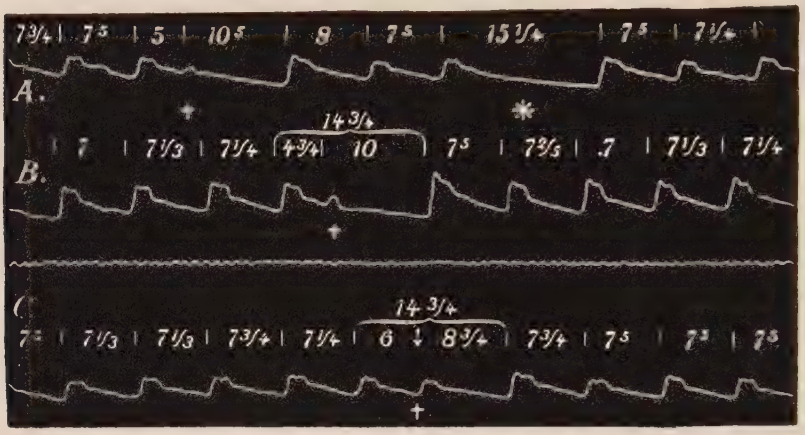


Fig. 90.—Radial tracing. Regular rhythm interrupted by extrasystoles. The numbers give the lengths of the pulse periods in one-tenth seconds. In A at * is a pure intermission, at † in A, B, and C, a smaller or larger pulse wave is visible in the intermission. The interval between the systole preceding the extrasystole and the systole following the compensatory pause is about the length of two normal pulse periods as the figures show. (Wenckebach, "Die Arhythmie," W. Englemann.)

Nodal Extrasystoles.—It has been shown experimentally that a stimulus applied to the auriculoventricular node is transmitted to both auricles and ventricles in about the same length of time and causes them to contract either at exactly the same time or almost simultaneously. The condition occurs clinically but is very rare. Its existence may be suspected from a tracing of the jugular pulse when there is a steep, large, premature *a* wave and when the *a-c* interval is reduced or absent or the *a* wave follows the *c* wave. The diagnosis can be safely made only with the electrocardiograph.

The Cause of Extrasystoles.—Extrasystoles are the most common irregularity. Probably few persons who attain to middle life have escaped having an occasional extrasystole at some time or other, although the majority have been quite unaware of its presence. Mackenzie found they could be detected in healthy individuals at all ages after infancy.

Rarely found before the age of twenty, they increase in frequency as age advances. They are most commonly found after middle age in persons who present evidence of arteriosclerosis (Hay), but Mackenzie says that his experience led him to expect them in every one over sixty years of age. Whether frequent and persistent extrasystoles are more common after forty years of age in patients who present definite signs and symptoms of heart disease than in those with normal hearts has not been settled. They are often met with in young nervous individuals. They may be produced in susceptible subjects by tobacco or alcohol. Digitalis induces extrasystoles in some people. I have seen one patient in whom extrasystoles occurred regularly after taking even small amounts of digitalis and they caused so much annoyance that she always insisted on giving up the drug after taking a few doses.

Symptoms and Signs.—Extrasystoles are more apt to occur when the heart-rate is slowing after slight acceleration. They are not common when the heart-rate is 100 per minute and very rare when it exceeds 120 (Lewis).

In the majority of people extrasystoles produce no discomfort. They are in fact unconscious of their presence. Some complain of distress during the compensatory pause following the extrasystole. In others the large beat following the extrasystole produces "palpitation" or a peculiar sensation that is often described as a "flopping" of the heart. It is as if it turned over. There may be an unpleasant fulness in the neck and throat following this forcible contraction. In neurotic persons extrasystoles are far more apt to cause distress than in those with more mental poise. When a number of extrasystoles follow each other at short intervals giddiness, sweating or coldness of the extremities may result or the output of blood from the heart may be so small as to lead to syncope, as with paroxysmal tachycardia, at times.

The extrasystole produces either a feeble pulse wave, or if the contraction were too weak to open the aortic valve, no beat at all at the wrist. The pulse following the long pause is unusually large. This is often well demonstrated in radial tracings.

A big wave in the jugular vein is often seen on inspection of the neck during a ventricular extrasystole and forms a prominent feature in many tracings of the venous pulse. The normal auricular contractions occurring before the premature ventricular contraction is completed cannot empty its contents into the ventricle and the blood is forced back into the superior vena cava forming the large wave seen in the jugular.

Diagnosis.—Extrasystoles can usually be easily recognized on auscultation of the heart. The regular recurrence of the heart-sounds of the

normal rhythm is interrupted by the sounds of the extrasystole occurring prematurely and followed by a long pause. Either two short, sharp sounds are heard or one feeble sound. If the ventricular contraction opens the aortic valve two sounds are heard. If the pressure in the ventricular cavity is too weak to open the aortic valve then only one sound is audible (frustrated contraction). If any sound is heard during an intermission of the pulse-rate it is almost conclusive evidence that the irregularity is due to an extrasystole.

On palpation of the apex region the premature contraction will often be found to produce an impulse of considerable force. If the extrasystole does not open the aortic valve it will transmit no pulse wave in the radial artery, yet the apex impulse may be strong. This is well shown in Fig. 91, in which there are two beats of the apex to one radial pulse.

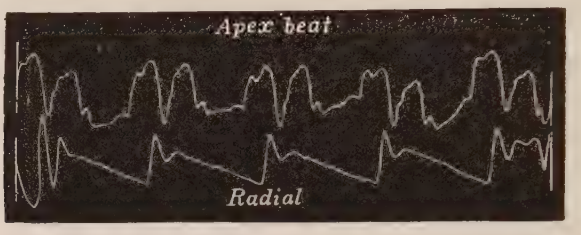


FIG. 91.—Simultaneous tracing of the apex-beat and of the radial pulse. There are two beats of the apex to one radial pulse. (Mackenzie, "Diseases of the Heart," Oxford University Press.)

Extrasystoles may occur singly or in a series. This depends on whether the irritable focus that forms the abnormal stimulus for contraction, is discharged once or repeatedly. If an extrasystole occurs after every normal contraction two pulse waves are usually found followed by a long pause. This gives rise to pulsus bigeminus. The pulsus trigeminus is due to a normal beat followed by two extrasystoles or by two normal beats followed by one extrasystole. This is separated from the next series of three beats by a long pause. If the extrasystoles are frequent and do not follow the normal beats in an orderly sequence the pulse will be very irregular. Such a pulse resembles closely that of auricular fibrillation. In attempting to distinguish between the two there are two simple aids to diagnosis. First—The rate of the pulse. An irregular pulse above 110 per minute is rarely due to extrasystoles, while the pulse of auricular fibrillation is often very rapid. Second—The effect of exercise. Increasing the pulse-rate by exercise removes an irregularity due to extrasystoles, but accentuates that due to auricular fibrillation.

To distinguish whether extrasystoles arise in the auricle or ventricle is not of great clinical importance. To do so requires the aid of instrumental methods. Simultaneous tracings of the waves due to auricular contraction in the jugular pulse and of the ventricular contractions in the arterial pulse enable the point of origin of the premature contractions to be determined. The electrocardiogram is of great value in the accurate analysis of extrasystoles.

TACHYCARDIA WITH A PATHOLOGICAL RHYTHM

Paroxysmal Tachycardia

The impulse arises in a new and pathological center in some part of the myocardium, usually the auricle. Paroxysmal tachycardia may be

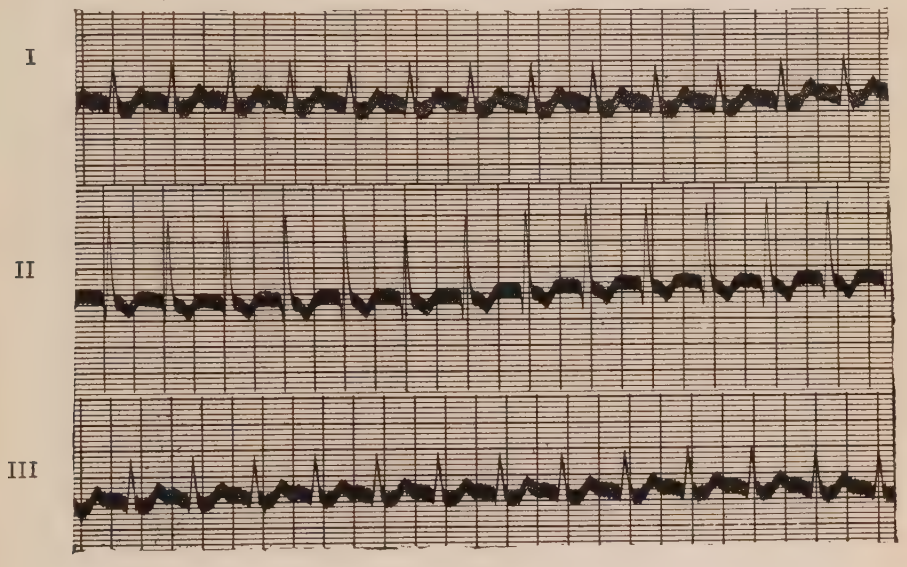


FIG. 92.—Auricular paroxysmal tachycardia. (Dr. Paul D. White.)

defined "as a condition in which from time to time the normal mechanism is interrupted by a series of rapid and regular beats, varying in rate between 100 and 220 per minute, the series starting and ending quite abruptly" (Lewis). (Fig. 92.) The fixing of the upper limit at 220 is rather arbitrary, but in clinical classification it is helpful. If a tachycardia with a pathological rhythm has an auricular rate above 220 it is regarded by Lewis as auricular flutter which will be described later. The form of tachycardia due to auricular fibrillation will also be discussed in another place.

The paroxysms are essentially a regular series of extrasystoles. (Fig. 93.) The whole heart is involved and the ventricle responds to every stimulus received from the auricle. The usual heart rate in a paroxysm is 160 to 200 per minute. There is a rare form in which the impulse arises in the ventricle. (Fig. 94.)

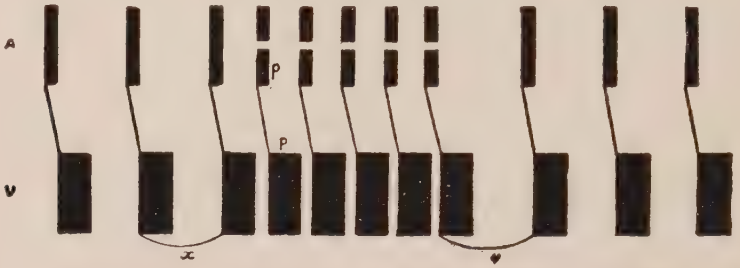


FIG. 93.—A diagrammatic representation of a short paroxysm of tachycardia arising in the auricle. The abnormal auricular beats are broken in their centers. Each yields a ventricular response. The first abnormal beat occupies the same position in relation to preceding events as does that of Fig. 87. The short paroxysm ends in a cycle *y*. *y* is longer than *x*. (Lewis, "Clinical Disorders of the Heart Beat," Shaw and Sons.)

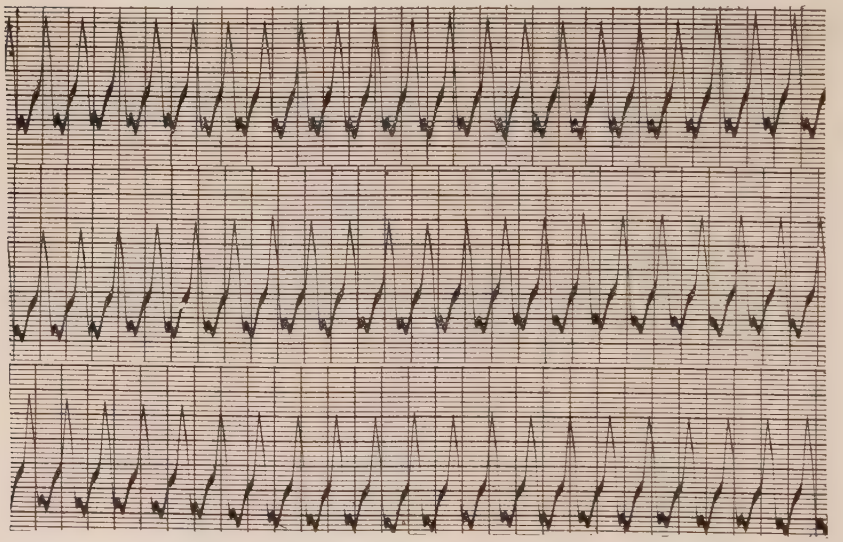


FIG. 94.—Ventricular paroxysmal tachycardia. (Dr. Paul D. White.)

Paroxysms may last from a few seconds to a fortnight. The usual duration is several hours. Attacks lasting more than twenty-four hours are uncommon in my experience.

A regular heart-rate of 160 or more in an adult should always suggest

to the observer the probability that he is dealing with a case of paroxysmal tachycardia.[1] The paroxysm begins suddenly and without warning and the decrease in rate at the end of an attack is equally abrupt. This sudden onset and offset are a great aid in the diagnosis. If the beginning of the seizure was not observed careful inquiry will reveal usually that the patient was conscious of the abrupt onset of the rapid heart action. The heart-sounds are usually fairly loud and similar in character having a tic-tac quality. Murmurs previously present usually disappear during the paroxysm. The heart-rate is not slowed when the patient changes from the erect to the recumbent position and this is a diagnostic sign of great importance. When a tachycardia is due to an acceleration of the normal

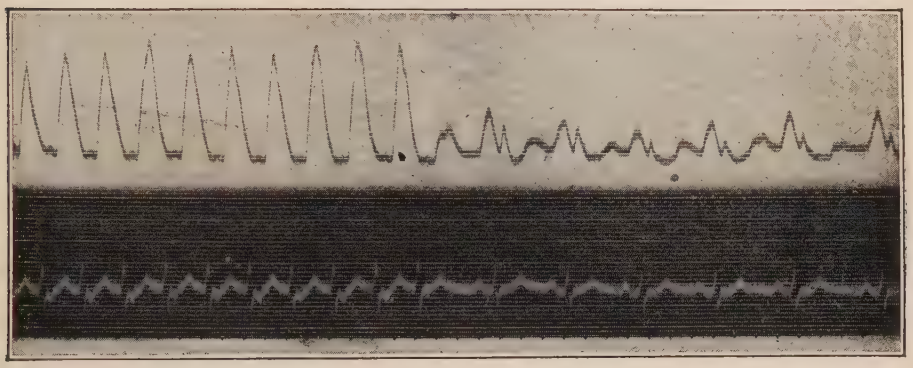


Fig. 95.—"Paroxysmal tachycardia with transition to a normal rhythm. The upper tracing is the record of the jugular pulse taken by Frank's optical method. The lower tracing is an electrocardiogram. Note that during the abnormal rhythm the inverted *P* falls directly after *R* of the preceding heart cycle and that since the auricle contracts during the preceding ventricular systole, it causes a single large venous wave. The last *R* of the tachycardia is not followed by an inverted *P*. The next auricular contraction originates in the sinus region (upright *P*) and thereafter the rhythm is normal." (Hewlett, "Pathological Physiology of Internal Diseases, D. Appleton and Company.)

sinus rhythm as in tuberculosis or exophthalmic goitre there may be a marked change in the rate with change in posture.

A prominent and palpable pulsation of the veins of the neck often occurs in paroxysmal tachycardia which may be "almost aneurysmal in force" (Lewis). A curve of the venous pulse frequently shows two waves instead of three, *a* and *c* forming a single big wave. This is characteristic. When the rate is very rapid there may be some delay in the conduction of the impulse from the auricles to the ventricles. This delay together with the rapid heart action may result in a contraction of the auricle be-

[1] When a tachycardia is suspected the rate at the cardiac apex should always be determined, as the heartbeats may be poorly transmitted to the wrist.

fore the preceding systole of the ventricle is completed, and the blood unable to enter the ventricle freely is in part thrown back into the great veins. This produces a striking change in the venous pulse. It is however of rare occurrence. (See Fig. 95.)

The rapid heart action reduces the cardiac reserve and the signs of cardiac insufficiency become more marked the longer the paroxysm and the more rapid the heart. The heart may dilate rapidly as a result of this strain. Venous engorgement increases. Congestion of the lungs is indicated by cough, accompanied by frothy or even bloodstained sputum and dullness at the bases with moist râles. The initial pallor increases and cyanosis may develop. The liver swells and the abdominal muscles may become rigid. There is pain and tenderness below the right costal margin. Hyperalgesia may be present over a wide area of the chest. Anginal pains or fatigue pains, as White prefers to term them, are common in prolonged seizures and may radiate down the left arm. With the cessation of the paroxysm the dilatation of the heart and signs of venous congestion disappear speedily. Polyuria may occur during recovery from the attack.

In most of the cases I have seen the attacks have been of short duration and the physical signs described above have been absent. The patients have usually complained only of palpitation, discomfort in the cardiac region and apprehension. The seizures may recur for many years without any evidence of heart disease being demonstrable in the intervals between the attacks.

AURICULAR FLUTTER

Auricular flutter is a condition in which the auricles contract at a rate of from 220 to 350 per minute. The usual rates are from 260 to 320 per minute and the usual ventricular rates are from 130 to 160, or half the auricular (Lewis).

Normally the contraction wave starts in the sinus node and spreads uniformally over the auricle, much as the wave spreads on the surface when a pebble is thrown into still water. The auricle remains at rest after a normal contraction until the next impulse is discharged from the sinus node. In auricular flutter there is a single and continuous wave in the auricle which moves in a circle. This is called a circus or continuous movement. The principle of circus movement is explained in Fig. 96. If the muscle ring is stimulated at x in Diagram A it will move only in a counterclockwise fashion if the tissue in the white zone to the left of x is in a refractory phase which blocks the contraction in the opposite

direction. When each position of tissue is excited the local contraction lasts, let us say, 0.1 second, as shown in Diagram B. During this phase the muscle is refractory. If the wave of contraction requires more than 0.1 second to complete the circle it will find each muscle fraction excitable again and the wave will continue to move about the circus path (Wig-

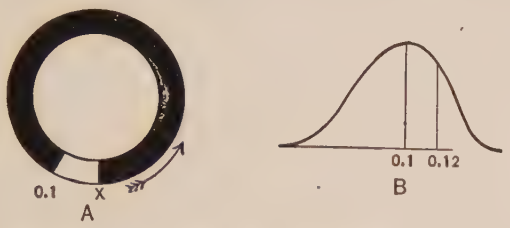


FIG. 96.—"Two diagrams illustrating the mechanisms of circus movements. A ring of muscle A, is stimulated at x at a time when a block exists just to the left of the point stimulated—the white portion of the circle. As a result of the block the impulse spreads in a counter-clockwise fashion only. As each portion of tissue is excited a local excitation and contraction result, lasting 0.1 second, represented in B. During this phase the muscle is refractory. If the rate of conduction around the circle is less than 0.1 second it will strike the muscle at x in the refractory phase and fail to produce a contraction. If on the other hand the impulse requires more than 0.1 second to travel around the circle it will find the muscle at x responsive again and will institute a circus movement. B, assumed contraction curve of any fraction of the ring shown in A, e.g. of x." (Wiggers, "Modern Aspects of the Circulation in Health and Disease," Lea and Febiger.)

gers). From this central or mother path centrifugal excitation waves pass to the more peripheral parts of the auricle. Fig. 97 shows that it may reach the left auricular appendage at the same time that it returns to the inferior vena cava. "The auricles are driven in merciless fashion and the reins of control, the inhibitory nerves, are powerless. The auricle has veritably seized the bit with its teeth. The ventricle, shielded from the whip by the auriculoventricular bundle, lags behind" (Lewis).

Rarely do the ventricles keep pace with the auricles. When they do respond to each impulse from the auricles it is only for a short time. The ventricular rate then becomes so rapid, 220 or more per minute, that the blood received from the auricles and thrown into the aorta during the very short cardiac cycles is so inadequate that loss of consciousness may result. Some degree of heart-block is almost always present. The usual ratio is 2:1, (Fig. 98) but all grades may develop, even to complete dissociation. When the ratio is changing owing to varying conductivity of the auriculoventricular bundle, the pulse becomes irregular. With the higher degrees of block, 3:1, 4:1 ratios, the ventricular rate and the pulse-rate may be normal and regular. No evidence may then be present on physical examination of this disturbance of the cardiac

mechanism. Occasionally faint rapid sounds of auricular origin have been heard over the heart. It was these sounds heard at such an unusual rate that led Cohn to take electrocardiograms of the first case in which he made the diagnosis. More frequently the auricular movements produce very rapid pulsation in the veins of the neck. These may be recorded in graphic records. They are beautifully shown in the tracing which Mackenzie made in a notable case and one of the first to be recognized. It was studied and reported by Hertz and Goodhart and was described

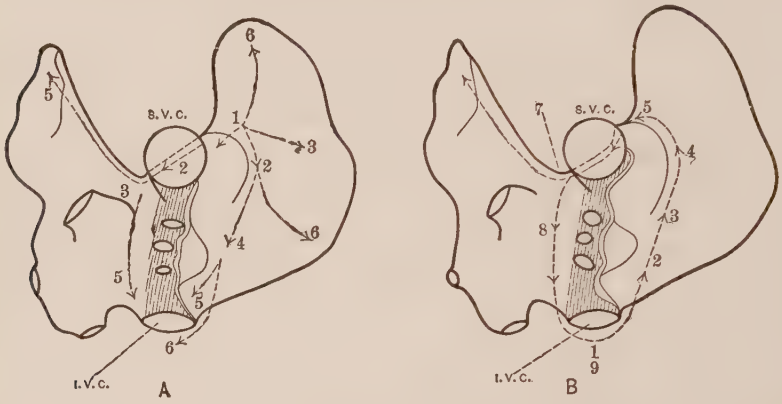


FIG. 97.—"Diagram of posterior portion of the auricles. A, showing order of excitation when impulse spreads normally from S–A node to right and left auricles. B, showing order of excitation in circus movements during flutter. In flutter the earliest excitation wave often begins at the inferior vena cava, 1, instead of the S–A node as normally. The impulse travels up the entire length of the tenia 2–5, moves around the superior vena cava across the interauricular band, 6–7, to the left auricle. It then passes downward, 8, and back to its place of origin, 9, at the inferior vena cava, where it reënters the circuit and establishes a circus movement. From this central or mother path excitation waves pass to the more peripheral tissue of the auricle. Thus as shown in B, the impulse may reach the left auricular appendix at the same time, 9, that it returns to the inferior vena cava." (After Lewis; Wiggers, "Modern Aspects of the Circulation in Health and Disease," Lea and Febiger.)

in a paper entitled "The Speed Limit of the Human Heart." This was before auricular flutter was recognized as a clinical entity. Polygrams in cases of flutter are often difficult of interpretation. Electrocardiograms are usually necessary to establish definitely the diagnosis. In them the form of the auricular waves is characteristic.

The radial pulse will be small if the rate is rapid. It may be regular as already stated or so irregular as to resemble the pulse in auricular fibrillation. In flutter the pulse-rate is not affected by rest, exercise or

emotion. The persistence of a steady pulse-rate of from 120 to 136 should always arouse the suspicion that the tachycardia is due to flutter, and lead to a thorough examination including an electrocardiogram. Direct pressure on the vagus or indirect vagus action by firm ocular pressure often slows the pulse. The vagus does this by acting on the auriculo-ventricular node increasing the grade of heart-block. It does not affect the auricular rate. Digitalis also has a striking effect in slowing the pulse in flutter. A tachycardia which has been suddenly slowed by digitalis is almost certainly auricular flutter. Flutter occurs in paroxyms of varying duration, which may closely resemble simple paroxysmal tachycardia. They may often be differentiated as Lewis points out by the pulse-rate. In paroxysmal tachycardia pulse-rates of from 180 to 220

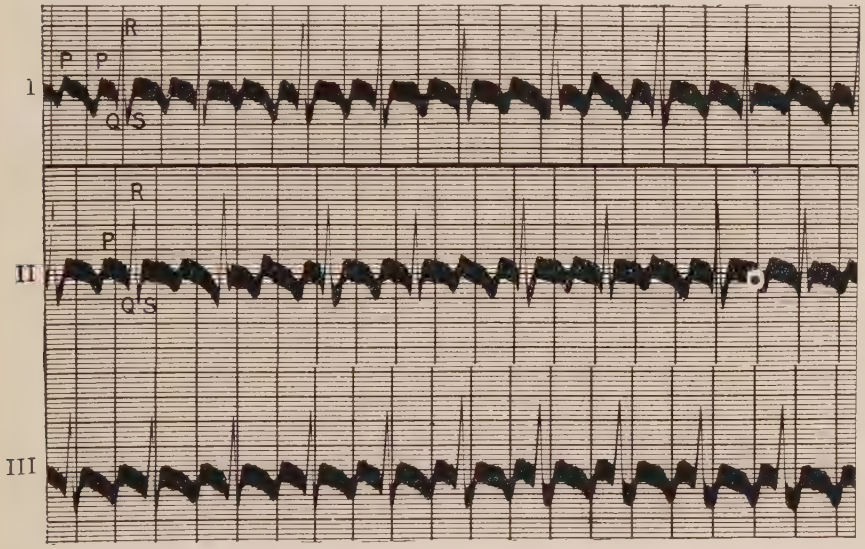


Fig. 98.—Auricular flutter. A 2:1 heart-block is present. (Dr. Paul D. White.)

are common. In flutter such ventricular rates are not common. The lowest ventricular rate in flutter without block is 220. Then the ventricle responds to every impulse reaching it from the auricle. The highest rate in a 2:1 block is about 190.

Flutter may persist for months or years. If the heart muscle is fairly strong the condition may produce no signs of congestive heart weakness even when a rapid ventricular rate has continued for years.

It is one of the rarer disorders of the cardiac mechanism. Although it may occur at any age it is much more common in the elderly.

AURICULAR FIBRILLATION

Although related to auricular flutter, which is a rare condition, auricular fibrillation is the most common irregularity to be found in the hospital wards. It is also known as *arrhythmia perpetua*. This is a poor name for this irregularity for although often permanent it does occur also in short paroxysms. Auricular fibrillation was the cause of the delirium cordis described by the older writers.

It may be defined as the condition in which the contractions of the auricle are replaced by an irregular circus movement which sends rapid and irregular impulses to the ventricles. It is characterized clinically by a pulse that is irregular in force and absolutely irregular in frequency. It is related to flutter, but instead of the impulse moving as a uniform wave, a circus, which produces a regularly beating auricle, the impulse in fibrillation is so frequently and so irregularly blocked in its course that it travels in an irregular sinuous path. Similar barriers of refractory muscle block the centrifugal waves that are given off from the sinuous circus ring. The multiple and variable sinuous waves are represented diagrammatically in Fig. 99.

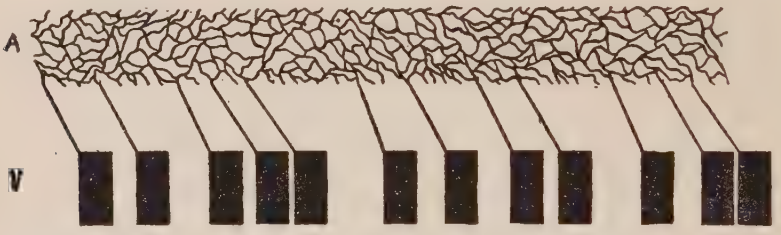


Fig. 99.—"A diagrammatic representation of auricular fibrillation. The fibres of the auricle do not contract coördinately. The contraction waves, which are multiple, follow varying and sinuous paths, colliding with each other or reëntering tracks through which they or their neighbours have previously passed. Occasional impulses leave the auricle at irregular intervals, and many of these proceed to the ventricle and stimulate it. Its action is therefore rapid and irregular." (Lewis, "Clinical Disorders of the Heart Beat," Shaw and Sons.)

The ventricles instead of receiving regular impulses from the auricles as they do normally are bombarded by haphazard impulses which escape from the 400 to 600 excitation waves that pass over the auricle per minute.

The recognition of auricular fibrillation is of the greatest importance clinically for it is in this condition that digitalis acts so beneficially. Digitalis slows the pulse-rate in auricular fibrillation and flutter, but rarely in other forms of tachycardia.

The higher the pulse-rate, the greater the irregularity in both the rate and the size of the pulse waves. In cases untreated with digitalis it is usual to find a rate of 120 or more over the heart. When the heart action is rapid many of the systoles are so weak that they fail to send an impulse to the wrist. As a result the rate may be normal (70 to 80) in the radial when it is 120 or more at the cardiac region. It is important to count the pulse at the apex as well as at the radial in all cases of auricular fibrillation or in those in which its existence is suspected. A record of the two rates should be kept on the patient's pulse chart. The differ-

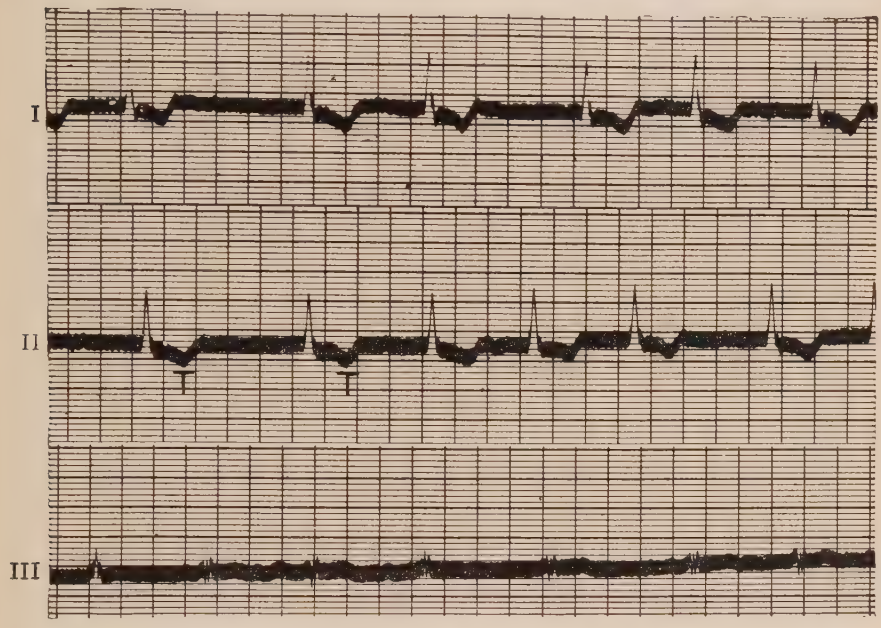


FIG. 100.—Auricular fibrillation. Note the absence of the P wave and the absolute irregularity of the heart-rate. Inverted T wave in the first and second leads due to digitalis action. (Dr. Paul D. White.)

ence between the rate at the heart and at the radial artery is termed the pulse deficit. As the patient improves the apex rate falls, the radial rate rises and thus the deficit is either lessened or removed.

The radial pulse tracing in addition to the irregularity in frequency and in the size of the pulse waves shows, unlike in other conditions, a lack of correspondence between the length of the diastolic pause and the size of the following pulse wave.

When the pulse is rapid the diagnosis is evident from the great irregularity in size and frequency of the beats. But when the pulse is slow

and the deficit has disappeared the rhythm may be sometimes mistaken for a regular one.

Systolic murmurs may disappear in auricular fibrillation when the heart-rate is rapid. They return when the heart-rate is slowed. With the onset of fibrillation the thrill and presystolic murmur of mitral stenosis disappear. This murmur is due to the contraction of the auricle and in auricular fibrillation the auricle no longer contracts. If attention is focused on the second sound at the apex a low-toned rumbling murmur will be heard beginning shortly after the second sound. If the rate is rapid it will fill the diastole and may be mistaken for a typical presystolic murmur. If the heart slows it will be found that a definite silent interval exists between the end of this murmur and the following first sound.

Diagnosis.—Auricular fibrillation can usually be recognized from the character of the pulse. Its diagnosis is aided by (1) attention to the rate, and (2) noting the effect of exercise on the irregularity.

An irregular ventricular rate above 120 is almost always due to auricular fibrillation. (Lewis.) If the rate is even more accelerated the probability is increased. If the symptoms of heart failure exist in association with a rapid pulse there can be little doubt of the nature of the irregularity.

If the heart-rate is slow and the irregularity slight moderate exercise will increase the irregularity in auricular fibrillation, but either lessen it or restore the regular rhythm if due to extrasystoles or heart-block. The slowing of the heart after exercise lessens an irregularity due to fibrillation, but increases an irregularity due to extrasystoles. There is a small group of cases of permanent auricular fibrillation in which the ventricular rate is slow, that is 50 or less, even when no digitalis has been taken, and in spite of increasing weakness of the heart this slow rate may continue.

Paroxysmal auricular fibrillation is much more common than was formerly thought. It is often unrecognized. Severe paroxysms have frequently been wrongly diagnosed as acute dilatation of the heart. (White.) If the ventricle is weak and the rate during a paroxysm rapid, dilatation may result, but dilatation never produces the irregularity. The idea although prevalent is fanciful. (Lewis.) The attacks may vary in length from a few seconds to many days, but short attacks are the rule. The sudden onset and offset of the rapid heart rate resemble those seen in paroxysmal tachycardia. The chief difference is that in paroxysmal fibrillation the pulse is irregular and in paroxysmal tachycardia it is regular. The duration of long attacks differs. Paroxysmal tachycardia has never persisted for more than two weeks. Paroxysmal auricular fibrilla-

tion has lasted much longer. Hay observed a case of twenty-eight days' duration. The signs of congestive heart weakness are as a rule more pronounced than in paroxysmal tachycardia. The ventricular rate is rapid (120–160). Often in long attacks symptoms are absent, but in these instances the ventricular rate is slower. Auricular fibrillation persisting over twenty-four hours is usually permanent.

This irregularity is rarely seen before the age of twenty. Its association with mitral stenosis is as common as its occurrence in aortic insufficiency is rare. Lewis puts it well when he says that "mitral stenosis and auricular fibrillation are bosom companions." In his series of 152 cases of auricular fibrillation 79 or 52 per cent had this valve lesion. It is also frequently found in myocardial degeneration (cardiosclerosis) of advanced life. In Willius's series of 500 cases observed in the Mayo Clinic 69 per cent were in myocardial disease and 31 per cent in chronic valvular disease.

In a group of 305 cases of auricular fibrillation reported by German observers 152 were associated with valvular disease, usually mitral stenosis, and 153 with myocardial disease.

I analyzed the records of 230 cases of heart disease seen in my own private practice. There were twenty-six cases of permanent auricular fibrillation. Twelve were associated with mitral stenosis, one diagnosed mitral insufficiency, but which I now believe was combined with mitral stenosis, thirteen cases of chronic myocardial disease (cardiosclerosis) and six cases of the paroxysmal type, all occurring in patients who had cardiosclerosis. From a consideration of all the collected cases the conclusion is justified that auricular fibrillation occurs with equal frequency in mitral stenosis and primary chronic myocardial disease (cardiosclerosis).

When the cardiac rhythm is normal there is usually present in cases of mitral stenosis a harsh crescendo murmur occurring just before the systole of the ventricle. This is the typical presystolic murmur of mitral stenosis. It is due to the contraction of the auricle which forces blood under increased pressure from the auricle through the narrowed mitral orifice into the ventricle. This murmur disappears with the onset of auricular fibrillation because the contraction of the auricle is replaced by a mere quivering of its wall. If there is a murmur present in early ventricular diastole before the development of auricular fibrillation it persists because it is not caused by the contraction of the auricle, but by the inrush of blood from the auricle into the ventricle through the narrowed orifice when the mitral valve opens. When the heart-rate in auricular fibrillation is rapid the murmur beginning in early diastole may

fill the entire diastolic interval up to the beginning of the next first sound. It then may simulate the true presystolic murmur. Often when the heart-rate in such a case is slowed by digitalis it is easy to make out a period of silence between the end of this murmur and the following first sound. This long diastolic murmur present when the heart rate is slow is frequently mistaken for that of aortic insufficiency (Mackenzie).

When a heart with fibrillating auricles weakens and signs of congestion develop there is usually a marked pulsation in the jugular veins which may not only be disturbing to the patient from the throbbing produced, but may be so marked as to attract the attention of the patient's friends. Tracings from this pulse show an absence of the *a* wave due to the auricular contraction, as Mackenzie discovered many years ago. The *v* wave due to the ventricular contraction is larger than normal and begins earlier in systole. To the type of venous pulse occurring in auricular fibrillation the name ventricular or positive venous pulse is applied, while the normal venous pulse is called the auricular or negative.

While auricular fibrillation is a serious disturbance of the cardiac mechanism it is viewed with much less apprehension than it was ten years ago. If the ventricular muscle is fairly healthy and strong, patients with this disorder may live for years and enjoy a fair measure of health provided the heart-rate is maintained at a normal rate by the continuous administration of digitalis.

HEART-BLOCK OR DISTURBANCE OF CONDUCTION

Heart-block is a delay or failure in the passage of an impulse through some part of the conducting system, usually the auriculoventricular bundle.

There may be (1) a sino-auricular block, (2) an auriculoventricular block, (3) an intraventricular block. By sino-auricular block is understood a delay or interruption in the passage of an impulse from the sinus node to the auricle. It should be distinguished from sinus bradycardia in which the impulse is slowly, but regularly formed and discharged from the sinus node. The term sino-auricular block should be limited to the rare instances of irregularity in which a ventricular silence is due to the failure of the sinus node to discharge an impulse. An auriculoventricular block may be partial when some impulses pass through the bundle of His, or complete when no impulse passes through the bundle. Intraventricular block is of two types: (a) bundle branch block due to complete blocking in one of the two large branches of the bundle of His that supply the right and left ventricle; (b) lesser grades due to widespread

blocking in the finer divisions or arborizations of the conducting system in the ventricular muscle or partial blocking of the main branches.

Etiology.—Heart-block in early life is usually the result of rheumatic infection. The largest group of cases occurs in the senile period and is due to degenerative changes in the bundle of His and its branches. A third group is caused by syphilis. In one-fourth of Lewis's cases that came to autopsy a gummatous lesion was found. Slight damage of the bundle, but insufficient to produce definite delay in the conduction time, is frequently brought to light by the administration of digitalis. In such cases heart-block promptly develops. It disappears when the drug is withdrawn. If the bundle is slightly diseased, pressure on the vagus may cause heart-block. Mackenzie reported a case in which a mild form of heart-block was produced by a reflex stimulation of the vagus on swallowing.

Different Forms of Heart-Block.—The relative frequency of the various forms of heart-block is given in the following table compiled by Dr. Paul D. White [2] from the cases observed at the Massachusetts General Hospital. Among 3219 cases electrocardiographed 263 or 8.1 per cent exhibited some form of heart-block.

	Cases	Per Cent
Auriculoventricular block		
Partial	129	4.0
Complete	27	0.8
Intraventricular block		
Bundle branch	41	1.2
Lesser degrees ("Aberration")	89	2.8
Sino-auricular block	11	0.3

Earliest Manifestations of Heart-Block.—The simplest form of heart-block is a delay in the propagation of the impulse from the auricle to the ventricle through the bundle of His. Normally the auricular systole precedes the ventricular systole by 0.2 second. The sequence is shown diagrammatically in Fig. 101. Simple prolongation of the interval between the auricular and ventricular contractions (the As–Vs interval) is illustrated in Fig. 102. The ventricular rate and rhythm are unchanged and this earliest manifestation of heart-block can rarely be recognized on the physical examination. Its identification requires the aid of polygrams or electrocardiograms. In these the waves representing the auricular and ventricular systoles are separated by a wider interval than normal.

[2] White, *Tr. Ass. Am. Physicians*, 1922, XXXVII, 277.

A widening of the interval may produce two signs that are useful in recognizing this form of heart-block. These are a reduplicated first sound and a reduplicated second sound. If either of these signs is present the evidence furnished by it should be confirmed by instrumental means before making a definite diagnosis. Normally the auricular contraction produces a faint sound, but it is masked by the much louder ventricular sound that follows it. When the As–Vs interval is slightly increased there may be a reduplication of the first sound. If the

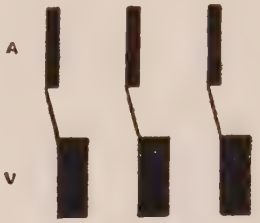


Fig. 101.—"A diagram representing the normal heart action. The auricle contracts first and transmits an impulse (represented by the oblique line) to the ventricle. The ventricle responds and commences to contract immediately at the cessation of auricular systole." (Lewis, "Clinical Disorders of the Heart Beat," Shaw and Sons.)

interval is more markedly widened the second sound may be reduplicated as the auricular contraction then falls in early diastole. In a case of mitral stenosis with regular heart action the presence of a thrill and murmur in early diastole may mean the existence of heart-block. (Cohn.) The explanation is simple. The thrill and murmur occur during auricular systole as a result of the blood being forced under higher pressure

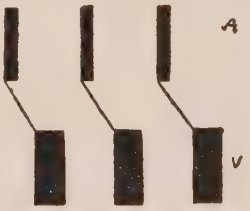


Fig. 102.—"A diagram illustrating the earliest stage of heart-block. An interval appears between the end of auricular and the commencement of ventricular systole. There is delay in the transmission of the impulse from auricle to ventricle (indicated by the increased obliquity of the line which joins the rectangles in the diagram)." (Lewis, "Clinical Disorders of the Heart Beat," Shaw and Sons.)

through the narrowed orifice by the contraction of the auricular muscle. Widening of the As–Vs interval increases the time between the auricular and ventricular systoles so that the auricular contraction occurs early in ventricular diastole.

Dropped Beats.—When the impulse from the auricle fails to pass through the bundle no ventricular contraction occurs and there will be a dropped beat in the radial pulse as a result. The ventricular rate before and after the missing contraction may be regular, but more often it is a little accelerated. Immediately after a blocked impulse the interval is usually lessened. It seems as if the rest permitted the bundle to propagate the impulse more rapidly for one or two cardiac cycles. On auscultation no sounds are heard when the beat is blocked. This ventricular silence distinguishes a dropped beat from an extrasystole which produces either a first sound alone or both sounds. Care should be exercised not to mistake a long pause of a sinus arrhythmia for a dropped beat. The long pause of a sinus arrhythmia it should be remembered is regularly associated with expiration.

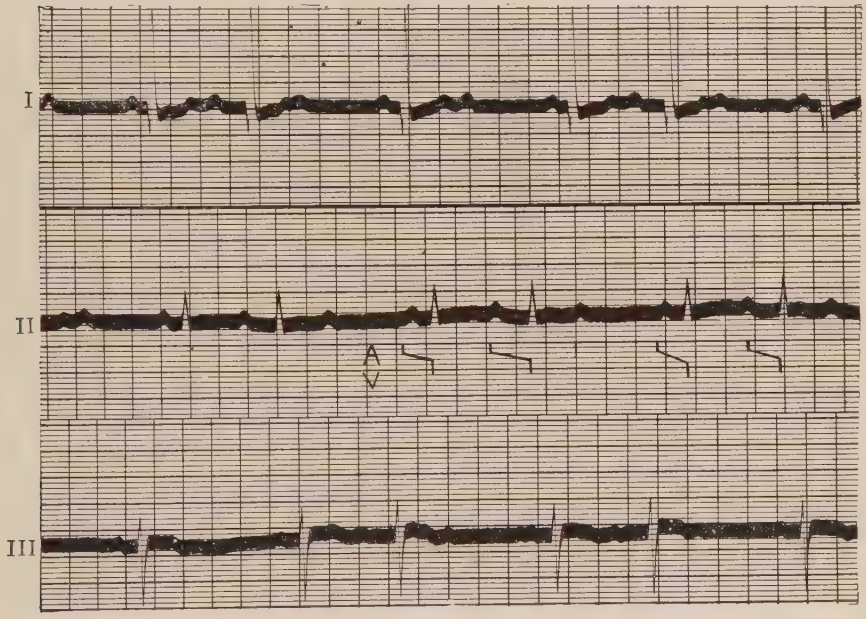


Fig. 103.—Partial auriculoventricular heart-block with dropped beats. Prolongation of the *P–R* interval with failure of the ventricles to respond to every second or third auricular impulse. (Dr. Paul D. White.)

Higher Grades of Block.—The blocking of impulses may occur with every other auricular contraction or it may be irregular. If alternate impulses from the auricle are blocked the ventricular rate is then one half of the auricular and the condition is called a 2:1 block. If every third or fourth impulse is blocked it is a 3:2 or a 4:3 block. A 2:1 block rarely persists for long. A sudden halving of the ventricular rate is

usually due to the development of a 2:1 block. A 3:1 and 4:1 block are very rare. If the ventricular rate is regular and between 40 and 50 to the minute a 2:1 block is to be suspected. Exercise in a case of 2:1 block may cause a sudden doubling of the heart rate. Subsequent rest results in an abrupt drop to the former rate.

All of these disturbances of conduction through the bundle, including the delay in the passage of an impulse, are embraced under the term partial heart-block.

Complete Block.—If no impulse passes to the ventricles from the auricle the ventricles beat independently. The auricles and ventricles are dissociated. The ventricle contracts in response to impulses that originate in its own pacemaker which is situated in the ventricular portion of His's bundle.

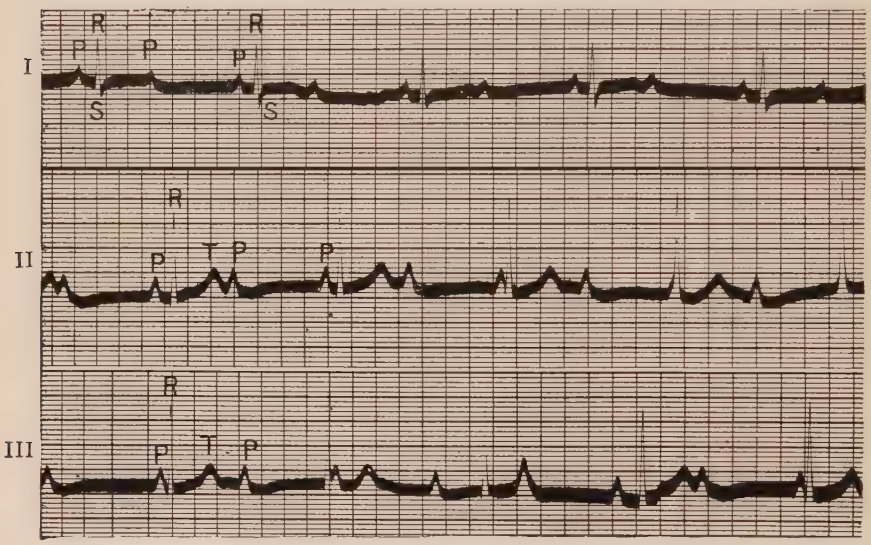


FIG. 104.—Complete auriculoventricular heart-block. The auricles and ventricles are beating regularly, but independently. The auricular rate is 86, the ventricular 42. (Dr. Paul D. White.)

In complete heart-block the rhythm is usually regular and the rate generally 35 or less. The rate is little if any affected by exercise. The radial pulse is usually full and forcible, and the systolic blood-pressure often 170–200 mm. if the heart muscle is strong.

Occasionally indications of the auricular systole are found on a tracing of the radial pulse. This is especially well shown in one made by Webster. (Fig. 105.) It is probably due to the impact of the auricle on the wall of the aorta.

On auscultation at the cardiac apex, but better at the lower end of the sternum, dull and distant sounds have sometimes been heard which are attributed to the auricular contractions. More frequently there is a definite and recurring change in the intensity of the heart-sounds independent of respiration, and this in cases of slow and regular heart action is a conclusive sign of complete heart-block. (Griffith.) An increase in the intensity of the first sound is due to the simultaneous contraction of the auricle and ventricle. In complete heart-block rapid pulsations in the jugular vein are often seen and a polygraphic tracing shows that small waves due to the auricular contractions (*a* waves), occur in the intervals between the ventricular contractions (*c* waves) and without relation to them. It should be remembered that the normal venous pulse in the neck consists of three waves to one in the radial pulse. The waves, however, are close together and the middle one of the three,

FIG. 105.—From Webster's case of heart-block showing in the falling line of the radial tracing a series of interruptions, (*a*) due to the left auricle. (Mackenzie, "Diseases of the Heart," Oxford University Press.)

the *c* wave, is due to the ventricular contraction. A wave in the jugular pulse occurring in the long ventricular pause is definite evidence of heart-block. A slow pulse due to extrasystoles that do not reach the wrist may be mistaken for heart-block if one neglects to listen carefully over the heart. There is usually in complete heart-block a variation in the size of the jugular pulse waves independent of respiration. Associated with a regular heart rhythm this waxing and waning of the jugular pulse is characteristic of heart-block. The larger waves occur when the auricle and ventricle contract at the same time. As the blood cannot enter the right ventricle it is forced back into the great veins and a large wave results.

Stokes-Adams Syndrome.—This rare clinical condition characterized by attacks of fainting or of convulsive seizure is associated with a very slow pulse due to heart-block. The attacks may occur either with partial heart-block of high degree or complete heart-block during periods of asystole. The ventricular rate is usually 30 or less. It may be as low as 7 or 10 to the minute, but this is very rare. Cessation of ventricular activity for from 3 to 7 seconds may produce dizziness or faintness or temporary loss of consciousness. If the asystole lasts 15 seconds or longer

convulsive movements occur. These are usually limited to the face and arms. Lack of adequate blood supply to the brain due to the long ventricular pauses is the cause of the loss of consciousness and epileptiform seizures.

Lewis and Cotton described attacks of faintness or unconsciousness due to emotional causes in soldiers with "irritable hearts" in which the pulse fell to 20 or 40 beats per minute accompanied by an independent fall of blood-pressure. This form of bradycardia is due to increased vagus action on the impulse formation in the sinus node, but not related to respiration. It is then a form of "sino-auricular block" and hence auricles as well as ventricles are slowed. Lewis believes this vagal disturbance is

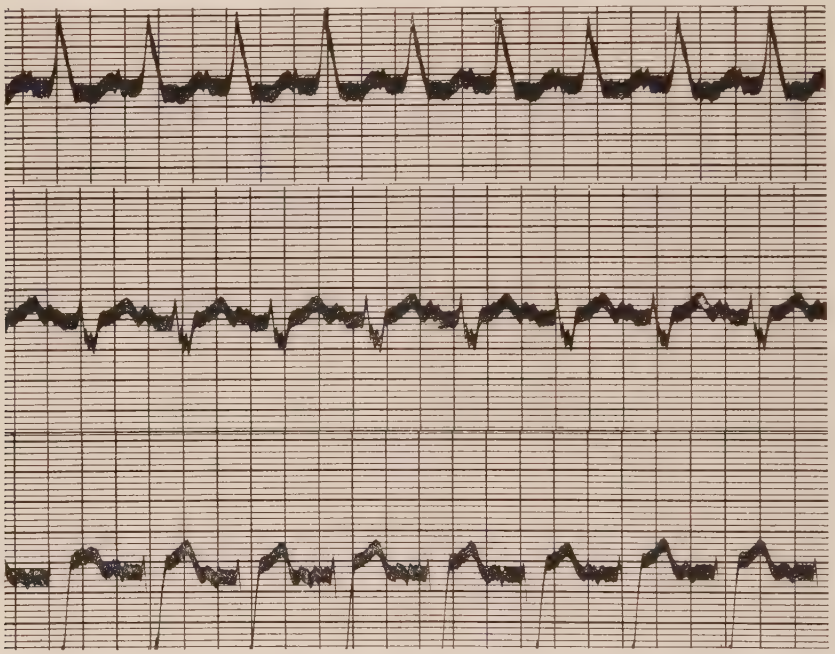


FIG. 106.—Right bundle branch block. (Dr. Paul D. White.)

the commonest cause of fainting attacks. It should never be confused with the Stokes-Adams syndrome.

Intraventricular Block.—Intraventricular block can be recognized only on electrocardiographic examination. It gives rise to a lengthening of the *Q-R-S* complex with changes in contour and amplitude. (See Fig. 106.) White's statistics show that it is almost as common as auriculoventricular block. Its recognition is important as its existence indicates profound changes in the muscle of the ventricle. In the severer

cases the right branch of the auriculoventricular conducting bundle is blocked. The left branch is rarely involved. (Fig. 107.) The *bundle branch type* occurs chiefly in old people as a result of arteriosclerosis. It is often associated with hypertension. The seriousness of the condition is shown by the fact that 23 of 36 cases in White's series died within a year of the discovery of the block. The lesser degrees of intraventricular block (*arborization block*) are twice as common as the bundle branch type. They also carry with them a grave prognosis as 36 per cent of White's cases died within six months of the time the condition was diagnosed. Although usually the result of senile changes in the heart they may occur in young people as a result of rheumatic infection.

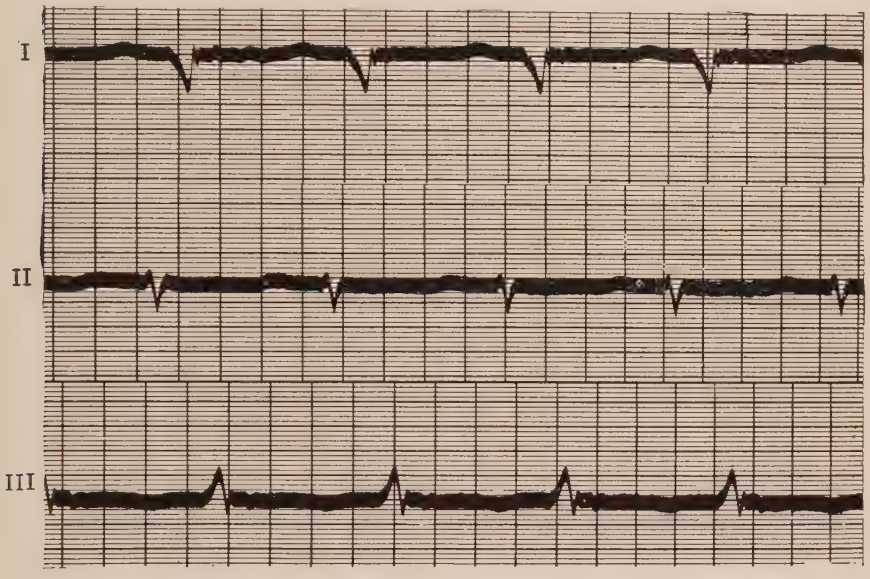


FIG. 107.—Left bundle branch block. (Med. Clin. N. A., III, Dr. Paul D. White.)

ALTERNATION OF THE PULSE

This is an alternation in the size of the pulse-beats without any change in the rhythm. The pulsus alternans was discovered long ago by Traube (1872)[3]. He noticed that the pulse waves in the radial artery while regular in time alternated in size. The condition is due to the expulsion by the left ventricle of larger and smaller amounts of blood at alternate contractions. (Fig. 108.)

It has often been confused with the pulsus bigeminus due to an extra-

[3] Traube, *Berl. klin. Wchnschr.* 1872, IX, 185.

systole occurring after each normal contraction. A radial tracing shows the difference between the two conditions. The small beat of the extrasystole is premature and is followed by a compensatory pause. As a result the longer pause follows the smaller beat. In pulsus alternans the interval between the beats is either equal or if there be a slightly longer pause it follows the larger beat. This may be due to a longer isometric period with the feebler ventricular contraction as Straub suggests or to a delay in the transmission of the smaller pulse wave to the wrist.

Alternation, unlike the other forms of cardiac disorders that have been described, is a disturbance of the function of contractility and not of conduction. The solution of its origin is a problem in cardiac dynamics. The exact nature of the disturbance is not clear. It is most likely that the larger beats result from the contraction of more muscle fibres of the ventricle than are active in the production of the smaller beats. On the other hand it is possible that all the muscle fibres of the ventricle are involved and that they contract with less force during the weaker systole than during the alternate stronger systole. Straub [4] holds that the es-

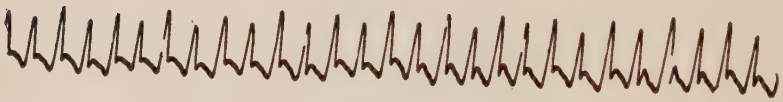


Fig. 108.—Pulsus alternans. (Dr. Paul D. White.)

sential feature in the physiology of its production is a deficient fall in the intraventricular pressure at the time the ventricle begins its contraction. The contractions that produce the weaker beats are slower, have a longer isometric period and propel less blood into the aorta. Rapid heart action and wide variation of pressure favor the occurrence of alternate weak and strong contractions.

There is rarely an electrical alternation that has been studied in electrocardiograms. It is distinct from the alternation in contractility. The mechanical alternation produces no change in the electrocardiograms. If the two forms of alternations chance to coexist in the same heart their independence of each other may be evident. The tall wave of the electrocardiogram (*R*- wave or *T*-wave) may correspond to the small pulse in the radial tracing or vice versa (Cohn).

Alternation has been produced experimentally by the injection of vari-

[4] Straub, *Deutsches Arch. f. klin. Med., Leipz.*, CXXIII, 1917, 403.

ous poisons. I have observed its development in rabbits after a fatal dose of diphtheria toxin.

Occurrence of Alternation.—It may be present when the ventricular rate is very rapid as in paroxysmal tachycardia or auricular flutter and disappears with the cessation of the attack. It then simply indicates that an excessive demand is being made on a heart muscle. Occurring as a result of tachycardia it does not indicate a weak heart muscle, in fact it has no clinical significance.

The existence of pulsus alternans when the rate is normal is conclusive evidence of a damaged myocardium.

There are two forms of pulsus alternans that should be recognized. First, the constant pulsus alternans. Fifteen of seventy cases studied by White were of this type. Second, alternation occurring for a few cardiac cycles after an extrasystole. Of White's fifty-five cases in this group, fifteen showed marked alternation, and forty only slight alternation. The premature contraction brings to light the hidden weakness of contractility.

Pulsus alternans is frequently associated with hypertensive disease and cardiosclerosis. It is relatively rare in young persons. No less than 62 per cent of White's cases were above the age of 50. It is a far more common condition than is generally taught. White found it present in 51 per cent of the patients with a blood-pressure constantly above 160 mm. Hg. who did not have auricular fibrillation.

Diagnosis.—Rarely can pulsus alternans be recognized by palpating the pulse, because the difference in the size of the pulse waves is usually too slight to be detected by the examiner's fingers. It produces no change in the heart-sounds.

The persistent alternation can often be discovered during a blood-pressure examination as Herrick pointed out. After the maximum blood-pressure has been determined drop the pressure a few millimeters. If alternation exists the sounds heard will be exactly half the heart-rate as the high pressure in the cuff prevents the alternate weak beats from passing through. In one of my cases alternation was detected in this way that could not be demonstrated in the radial tracing.

The more common form of alternation that occurs only after a single extrasystole can only be recognized in a tracing of the arterial pulse. The importance of pulsus alternans in the diagnosis of myocardial degeneration and in prognosis justifies the routine employment of the sphygmograph in every case of cardiac insufficiency. By the routine use of the sphygmograph in the wards and out-patient department of the Massachusetts General Hospital, White collected seventy-one cases of alterna-

tion in a period of eight months. Lewis urges systematic search for this important sign in all cases of angina pectoris, of high blood-pressure, of suspected heart disease or known kidney disease and of frequent extrasystoles in all elderly patients. He points out that the patient should remain standing while the arterial tracing is made as extrasystoles are more frequent in this position. It is well to make the examination shortly after the arrival of the patient in the physician's consulting room as extrasystoles are more apt to occur before the effect of exertion or emotion has passed away. If no extrasystoles occur they may sometimes be produced by holding the breath.

Prognosis.—Continued alternation of the pulse is an ominous sign. Its presence is a sure indication that the end is near. In a few months after the discovery of this sign in most cases and within a few years in all cases death occurs. The longest duration of life I have seen was three years after pulsus alternans was detected. In the last two years of this period the patient was compelled to spend most of his time in bed owing to cardiac weakness. My other cases have died within a year. Less is known regarding the prognosis in the less developed form occurring only after extrasystoles. According to White's statistics the significance of this form of alternation if well marked is almost as grave as the continued form; forty-seven per cent of his patients dying within the year. Alternation never occurs as the sole sign of cardiac weakness. Dyspnea on slight exertion, cardiac asthma, angina pectoris and Cheyne-Stokes breathing are often found, singly or together, in association with alternation of the pulse.

PULSUS PARADOXUS

This is characterized by a diminution in the size of the pulse waves or their disappearance during inspiration. It should not be confused with sinus arrhythmia, but both conditions may occur simultaneously.

It was first observed by Griesinger, in mediastinopericarditis, and described more fully a little later by Kussmaul [5] who gave the name pulsus paradoxus. He applied this term he tells us partly because in his case there was a surprising lack of relation between the heart action and the arterial pulse, and partly because the pulse in spite of an apparent irregularity showed actually a regular recurrence of missing pulse waves or waves of smaller size. The term is a rather unfortunate one as there is really nothing paradoxical about the pulse.

[5] *Berl. klin. Wchnschr.* 1873, X, 433.

There are according to Wenckebach [6] three causes for the smaller size of the radial pulse in inspiration:

(I) an extrathoracic cause

(II) a dynamic cause

(III) a mechanical cause.

The first is a pseudoparadoxical pulse. It is due to compression of the subclavian artery between the first rib and the clavicle by the elevation of the thorax during inspiration.

In the second form a great increase in the negative pressure during inspiration leads to dilatation of the ventricles and aorta and to diminished filling of the systemic circulation. This form may occur according to Wenckebach under a variety of conditions including very deep and rapid inspirations, obstruction to entrance of air into the lungs, and great reduction of the surface area for respiration, as in large pleuritic effusions.

The third form is that observed by Kussmaul. During inspiration the blood flow to the heart is decreased by the pressure of mediastinal bands or other structures on the vena cava. At least this is the theory of Kussmaul and it has been generally accepted. The decreased inflow results in a decreased output from the left ventricle.

The paradoxical pulse has been observed in pericardial effusion. It has recently been produced experimentally by injecting fluid into the pericardial sac of animals breathing naturally with the thorax closed (Katz and Gauchat). When the pericardial sac was filled with fluid the heart was less affected by the respiratory changes in intrathoracic pressure. As the veins entering the heart were still subject to these variations in pressure the result was that when the intrathoracic pressure decreased during inspiration the blood accumulated in the veins and little or no blood entered the heart.

Usually the heart-sounds are well heard during inspiration, but I have observed a case in which both sounds were absent during inspiration although loud during expiration. The tracing in Fig. 109 is from this case. The sudden disappearance of the heart-sounds indicates that little or no blood entered the heart during inspiration. Retention of the blood in the lungs and the great veins is the probable explanation. The patient from whom the tracing was made had a mediastinal tumor. The radial tracing shows very small or absent pulse waves during inspiration. The upper curve was made from the second right interspace near the sternum. It shows a definite retraction of the chest wall at the onset of inspiration.

[6] Wenckebach, *Ztschr. f. klin. Med.,Berl.*, 1910, LXXI, 402. See also Wenckebach, "Die unregelmässige Herztätigkeit und ihre klinische Bedeutung," 1914, 215–223, for the best discussion of the paradoxical pulse.

The pulsus paradoxus due to a dynamic cause can be distinguished according to Wenckebach from that of mechanical origin by a difference in the pulse curves. In the dynamic form the first waves or all the waves during expiration are the largest, while the smallest occur during inspiration and those of medium size during the respiratory pause. In the mechanical form the pulse waves diminish during inspiration, increase during expiration and are largest during the respiratory pause. The pulse tracing reproduced in Fig. 109 from my patient with pulsus paradoxus is due to a dynamic cause if Wenckebach's view is correct because the largest waves occur early in expiration.

In pulsus paradoxus there is often a striking fall of the blood-pressure during inspiration. This is indicated in Fig 109 by the drop in the base line of the pulse waves.

Often in association with the mechanical form of pulsus paradoxus the jugular veins in chronic mediastinopericarditis exhibit a paradoxical be-

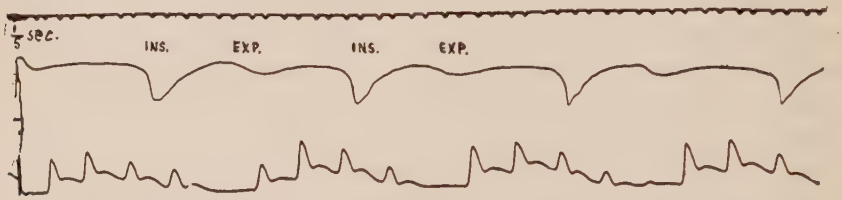


Fig. 109.—Pulsus paradoxus. Note the great irregularity in the size of the radial pulse waves. During inspiration the waves become very small or disappear. During expiration the waves attain their largest size. The fall of the base line during inspiration is due to the drop in the blood-pressure. Upper tracing records movement in second right intercostal space near sternum. At onset of inspiration there was a retraction of the chest wall at this point.

havior. Instead of collapsing in inspiration and filling during expiration as they do under normal conditions, they become distended during inspiration and are emptied during expiration. There was no overfilling of the veins in my case of pulsus paradoxus during inspiration. Nor was it to be expected, as in the dynamic form of the pulsus paradoxus an inspiratory collapse always occurs. (Schreiber, Wenckebach.)

A degree of pulsus paradoxus sufficient to attract attention in the routine examination of the pulse by palpation is rare. It may easily be produced normally by forced thoracic type of respiration, quiet abdominal breathing failing to show it. (Lewis.)

CHAPTER V

TESTING THE FUNCTIONAL POWER OF THE HEART

More important than an anatomical or an etiological diagnosis is the determination of the strength of the heart. It is with the functional diagnosis that the patient is chiefly concerned. He comes to the physician to ascertain if his heart is strong or weak. The reserve power of the heart is the measure of its strength and the limit of its accommodation. Mackenzie has emphasized in his writings the importance of determining in every case of heart disease the limitation of the field of the cardiac response to effort and he regards the patient's sensations as the best guide to the efficiency of the heart. When a person finds that exercise which he was accustomed to do with ease causes breathlessness, substernal distress, or a sense of exhaustion it is safe to conclude that his cardiac reserve is diminished, provided that a neurotic condition can be excluded as the cause of these subjective symptoms. To maintain an adequate circulation the heart must supply all the tissues with sufficient blood to meet their needs. The amount required by the skeletal muscles is greatly increased by exercise. The heart is forced to pump much more blood into the aorta against a higher pressure. To do this it must receive more blood from the venous channels. The increased volume of blood flowing into the ventricles from the auricles distends the muscle fibres and causes them to contract with greater force, and the increase in initial tension thus produced enables the ventricle to expel into the aorta all the blood it receives from the auricle. The height to which the initial tension rises is the measure of the reserve power. When an increase in the diastolic filling no longer leads to an increase in the systolic output the limit of the heart's accommodation, or in other words of its reserve power, has been reached.

Unfortunately no satisfactory clinical test has been devised for measuring either the work of the heart or the volume of the cardiac output. The patient's statements as contained in a properly taken history should be given great weight especially in the detection of beginning weakness of the heart. Often only by means of information obtained by skillful inquiry can it be determined whether cardiac symptoms such as pain in the cardiac region or dyspnea are due to organic disease of the heart muscle.

or are the expression of a psychic disturbance in a person whose heart is sound and strong. The symptoms reported by the patients are only his subjective impressions and may be vague and misleading. A trustworthy history depends upon the critical evaluation by the physician of the patient's statements in the light of the objective findings and of his knowledge of the patient's personality. (Romberg.) No rules can be given for taking an adequate history in a case of cardiac disease. To acquire skill in history taking is more difficult than to carry out properly the objective examination. The amount of effort required to produce discomfort should always be ascertained and recorded. Some patients are hypersensitive and are conscious of the slightest disturbance in cardiac rate or rhythm and of any change in depth or rapidity of respiration. Others are quite unaware of an alteration in breathing that is sufficiently marked to attract the attention of their companions. For this reason it is often important to include in the clinical record the observations of other members of the patient's family.

The examination of the heart itself reveals less evidence of cardiac weakness than that of other parts of the body particularly of the lungs and pulse. The development of dullness and râles at the bases of the lungs may be the first sign of the beginning cardiac failure. The alternating pulse is definite evidence of serious impairment of the function of contractibility.

The effect of some simple exercise should be carefully noted on the rate and depth of respiration, the force of the cardiac impulse, the heart-rate, the color of the lips and skin and the general aspect of the patient. Many attempts have been made to devise functional tests for determining the power of the heart. None of them has been entirely successful. Many are based on changes in the pulse and blood-pressure. The effect of exercise on pulse and blood-pressure is dependent, however, not only on the condition of the heart but also on the state of the nervous system, the power and condition of the skeletal muscles, and of the peripheral circulation, and there is no way of determining what part of the total effect is due to the heart.

Bearing in mind their limitations, something may be learned from simple exercise tests. That of Kuhn has been widely used, and the technic is simple. The patient bends his knees ten times in fifteen seconds. Immediately before and after this exertion, the pulse is counted for a quarter of a minute. The exercise normally increases the pulse rate during this period 4 to 9 beats, equal to an acceleration of 16 to 36 beats to the minute. After 45 seconds or at most a minute and a half the rate should drop to the previous level. Greater acceleration or

a longer continuation of the rapid heart-rate is taken as an indication of functional disturbance of the heart. Nervous persons with a labile pulse but a perfectly sound heart muscle will exhibit the same or a greater acceleration of the pulse than a patient with a weakened heart muscle, and herein lies the defect of this and similar tests.

The most practical test of cardiac power, is the vital capacity which gives a fairly trustworthy, although an indirect, measure of the reserve power of the heart. It has been discussed in a preceding chapter.

CHAPTER VI

THE CLASSIFICATION OF CARDIAC DISEASE

In every case of heart disease, a threefold diagnosis should be made. The first deals with etiology, the second with structural change and the third with the functional condition. All are important, but the anatomical diagnosis is less essential than the etiological or the physiological. Yet it was not many years ago that the diagnosis of structural change was the only one made in most cases (White). It is also important in each case to estimate as closely as possible the power of the heart; that is, its functional capacity or reserve power.

The classification given below is a combination, with some modifications, of one proposed by White and Myers and that of the New York Committee on Cardiac Clinics.[1]

Etiological Diagnosis

1. Congenital heart disease
2. Rheumatic infection
3. Diphtheria
4. Syphilis
5. Streptococcus viridans
6. Bacteria of sepsis (streptococcus hemolyticus, staphylococcus aureus, et al.)
7. Tuberculosis,—a frequent cause of pericarditis with effusion
8. Hyperthyroidism (the thyreotoxic heart)
9. Arteriosclerosis
10. Hypertension
11. Psychoneurosis

It should be stated whether the exciting cause is (A) active, or (B) inactive.

Anatomical Diagnosis

1. Hypertrophy of the heart
2. Ventricular preponderance

[1] *J. Am. M. Ass., Chicago,* 1921, LXXVII, 1414. *Boston M. & S. J.,* 1923, CLXXXIX, 765.

3. Dilatation of the heart (a) auricles, (b) ventricles
4. Infarction of the heart
5. Rupture of the heart
6. Acute myocarditis
7. Chronic myocarditis
8. Cardiosclerosis (the senile heart)
9. Acute endocarditis
10. Chronic valvular disease (chronic endocarditis)
    a. Aortic insufficiency
    b. Aortic stenosis
    c. Mitral insufficiency
    d. Mitral stenosis
    e. Pulmonic stenosis
    f. Tricuspid stenosis
11. Dilatation of valvular orifices (muscular insufficiency)
    a. Mitral insufficiency
    b. Tricuspid insufficiency
    c. Pulmonic insufficiency
12. Congenital abnormality. Specify lesion, if possible
13. Pericarditis
    a. Acute (fibrinous, serofibrinous)
    b. With effusion (serous)
    c. Purulent
14. Adherent pericarditis (chronic mediastinopericarditis; adherent pericardium)
15. Hydropericardium
16. Pneumopericardium
17. Acute aortitis
18. Chronic aortitis
19. Aneurism of the aorta. Specify affected part
20. Coronary thrombosis

Functional Condition (Physiological Diagnosis)

1. Cardiac insufficiency (cardiac weakness; heart failure)
   A. Congestive type. Chronic passive congestion of various organs, edema, cyanosis, distention of jugular veins
   B. Anginal type. Pain or pressure
2. Disordered heart action
   A. Extrasystoles (premature contractions): (a) auricular, (b) ventricular

B. Paroxysmal tachycardia: (a) auricular, (b) ventricular
C. Auricular flutter: (a) paroxysmal, (b) chronic
D. Auricular fibrillation; (a) paroxysmal, (b) chronic
E. Auriculoventricular heart-block
   a. Partial block
      1. Prolonged conduction time
      2. Dropped beats
   b. Complete block
F. Intraventricular block
G. Auriculoventricular rhythm and ventricular escape
H. Pulsus alternans

3. Hypertension
   a. Essential (primary)
   b. Associated with Bright's disease (secondary)

FUNCTIONAL CAPACITY (RESERVE POWER)

1. Patients with organic disease but able to carry on ordinary physical activity
2. Patients with organic disease, but unable to carry on ordinary physical activity (cardiac insufficiency)
   a. Activity slightly limited
   b. Activity greatly limited
3. Patients with organic disease, but unable to carry on any physical activity, i. e., who must remain in bed or in a chair (cardiac insufficiency).

CHAPTER VII

DIAGNOSIS OF DISEASES OF THE HEART VALVES AND MYOCARDIUM

Cardiac Insufficiency (Cardiac Weakness, Heart Failure)

If the heart is no longer able to supply adequately all the organs and tissues of the body with blood, subjective symptoms and physical signs of a disturbance in the circulation develop. These are due to an altered distribution of the blood. The lungs or the systemic venous system or both are overfilled with blood (chronic passive congestion). Cardiac insufficiency develops in the various forms of heart disease, when one or both of the ventricles weaken. This occurs in valvular disease, in acute myocarditis, in cardiosclerosis, in hypertensive heart disease, in pericarditis, in the thyroid heart, in heart strain, emphysema, and kyphoscoliosis.

The subjective symptoms resulting from cardiac insufficiency are breathlessness, at first usually only on exertion, attacks of nocturnal dyspnea (cardiac asthma), palpitation, pain in the cardiac region or behind the sternum, pain below the right costal margin due to the congestion of the liver.

The physical changes in the heart have already been described. The early objective signs of cardiac failure are dyspnea, cyanosis, congestive bronchitis due to chronic passive congestion of the lungs, swelling of the liver, distention of the jugular veins. The urine is scanty and high colored and often contains albumin. These alterations in the urine are the result of chronic passive congestion of the kidneys. Ascites may be a consequence of venous stasis in the abdominal organs. General edema often develops, usually beginning about the ankles or over the tibiae. Hydothorax or hydropericardium may occur owing to congestion in the pleural or pericardial veins. General edema may be absent and the congestion be limited to the pulmonary circulation. This is the case when the left ventricle weakens more than the right; when the failure of the right ventricle predominates so that the left is relatively strong there may be swelling of the liver and general edema with little dyspnea and no congestion of the lungs.

Acute Endocarditis, Acute Myocarditis and Acute Carditis

Most cases of so-called acute endocarditis are really instances of inflammation of the myocardium as well as of the heart valves. Often the three parts of the heart—endocardium, myocardium and pericardium—are all involved, but to a different extent. Hence the old term carditis is more accurate than endocarditis or myocarditis.

Rheumatic infection is the most common and most important cause of acute myocarditis. The disease is not always secondary to polyarthritis. It may follow a rheumatic pleurisy or tonsillitis, or develop as a primary rheumatic endocarditis. The causative microörganism of rheumatic fever is unknown. So-called septic endocarditis is due to infection with staphylococcus aureus, streptococcus viridans and more rarely pneumococcus, gonococcus and other bacteria. Acute myocarditis can occur as a complication in the course of various infectious diseases, especially diphtheria, scarlet fever, and typhoid fever.

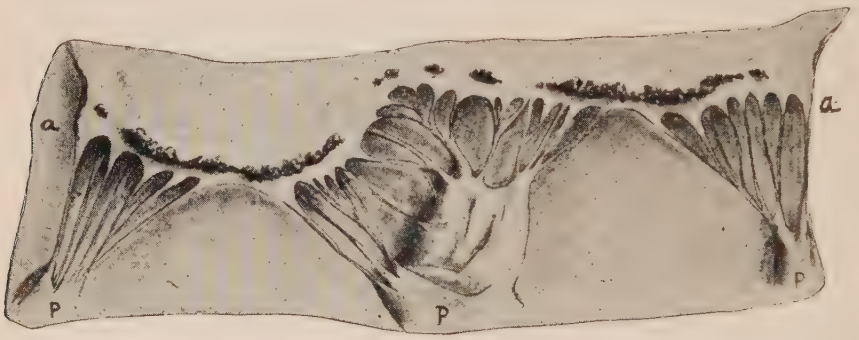


Fig. 110.—Acute endocarditis of the mitral valve. P P P Papillary muscles. The valve itself is not deformed, the distance from *a* to *a* measures 11 cm. On the surface of the valve near and parallel to the free margin is a series of thrombi. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," F. C. W. Vogel.)

The clinical picture of bacterial and rheumatic endomyocarditis is often that of a septicemia which may be mild or severe. Cardiac enlargement of one or both chambers may develop early in rheumatic heart disease but in the bacterial endocarditis it may not occur until the late stages.

In acute myocarditis the cardiac mechanism is often disturbed. A delay in conduction of the impulse through His's bundle is common in the acute rheumatic heart, and dropped beats may occur due to heart-block. The pulse is usually rapid, soft and small. The heart-sounds may be

clear or systolic murmurs be heard at the apex or less commonly at the base. They occur when the valves are intact as well as when they are ulcerated and the seat of vegetations. The murmurs are often due to involvement in the inflammatory process of the muscular ring that surrounds the auriculoventricular orifice. The presence of a systolic murmur is no evidence of endocarditis. A myocarditis can produce it just as well (Krehl).

The differentiation between endocarditis and myocarditis is very difficult at the onset of the disease and not important. The disturbance in the function of the heart is due to the changes in the heart muscle.

Aortic Stenosis

Aortic stenosis is the rarest of the left-sided valvular lesions. It occurred in only five per cent of the 1781 cases of valvular disease admitted to the Johns Hopkins Hospital between 1889 and 1908 (Hirschfelder). There were only six cases among 462 acquired valvular lesions examined in Romberg's clinic in Munich. In some series the percentage is higher. For example D. Gerhardt met with nineteen cases among three hundred of valvular disease, and fifteen of these were of the pure form, that is, uncomplicated with aortic insufficiency or other valvular lesion. Gerhardt's figures are exceptional. All authorities are agreed that aortic stenosis is seldom pure, but usually associated with aortic insufficiency. The disease is due to a thickening and fusing together of the aortic leaflets the result of chronic inflammation or atheromatous change. The thick deformed inelastic aortic cusps are rarely able to close the orifice completely at the end of diastole and hence some leakage of blood from the aorta into the ventricle usually occurs. This explains the frequent association of aortic insufficiency with aortic stenosis. It is in the high degrees of constriction, when the aortic opening is reduced to the diameter of a lead pencil or less, that pure aortic stenosis is most apt to occur. Then a minimal movement of the valves is able to close the orifice sufficiently to prevent regurgitation. The less the stenosis the greater the difficulty the deformed valves have in closing the opening.

Etiology.—Sclerotic changes in the valve and rheumatic fever are the usual causes of the lesion. Rheumatic fever is responsible for the group of cases in young people. Syphilis is the etiological agent third in importance. There is a congenital form of the disease but it is very rare (H. Vierordt).

Pathological Physiology.—Narrowing of the orifice throws increased

work on the left ventricle and it becomes hypertrophied. The increased resistance to the emptying of its contents is not great until the size of the orifice is reduced one-half. Greater reduction than this increases greatly the work of the left ventricle. Experimental studies show that the intraventricular pressure during systole and the duration of systole increases with the degree of stenosis. When the peripheral resistance was high it required a greater constriction to produce changes with cardiac dynamics than when the peripheral resistance was low. (de Heer.) (Fig. 112.) The increase in the length of systole is due to a lengthening of the ejection period. If a stenosis is produced there is a diminished systolic output and an increase of the volume of blood left in the ventricle at the end of systole. This produces a slight dilatation and this in turn an increased initial

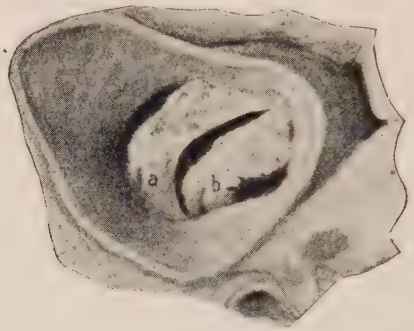


Fig. 111.—Stenosis of the aortic valve. The segments are much thickened and calcified. Two segments are fused together and form the mass a. The ostium is reduced to a small cleft. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathological Anatomie," F. C. W. Vogel.)

tension. According to the most recent view it is the increased initial tension that is the direct cause of the hypertrophy. In the clinical cases with their slow development it is possible that after hypertrophy of the muscle occurs there is no dilatation and no increase of initial tension (Romberg). It is certainly true that a dilatation sufficient to be demonstrated in the Roentgen examination may be absent if the heart muscle is strong. Aortic stenosis still furnishes the typical clinical example of cardiac hypertrophy without dilatation. In cases combined with aortic insufficiency, and after the onset of cardiac weakness in the pure form of the disease, dilatation of the left ventricle occurs. Increased weakness of the left ventricle leads to hypertrophy and dilatation of the left auricle and right ventricle.

Inspection.—The cardiac impulse is usually heaving. It may be in its normal position or dislocated a short distance outward and downward.

Palpation.—An intense thrill is felt in the second right interspace near

the sternum in a large proportion of the typical cases. Dr. Osler always cautioned his students to be wary of making the diagnosis in the absence of this thrill. Occasionally the thrill can be felt over the carotid arteries. Hypertrophy of the heart always occurs and in most cases this will be indicated by a definite heave of the apex impulse, a "well defined and deliberate pulse of no great violence." (Broadbent.)

Percussion.—The cardiac outline may be normal or increased to the

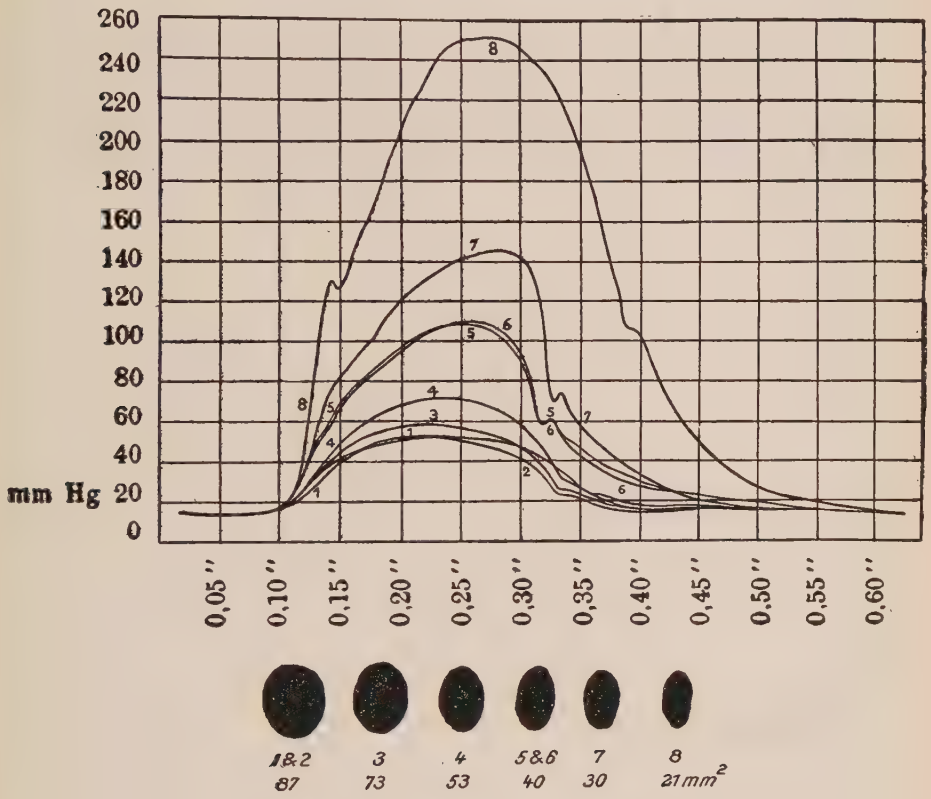


FIG. 112.—Pressure curve in the left ventricle with increasing constriction of the aorta. The degree of stenosis is indicated by the numbers 1 to 8. Below is a series of cross-sections of the aorta as it becomes more and more narrow. The lowest row of numbers gives the area of the opening in square millimeters. The oval at the left (numbers 1 and 2– 87 sq. mm) represents the normal size of the aorta of the dog from which the pressure curves were obtained. (After de Heer; Lüdke and Schlayer, "Lehrbuch der pathologischen Physiologie," J. A. Barth.)

left and downward. The shape of the orthodiagram is similar to that in aortic insufficiency, but smaller. The width of the aorta is not increased as in aortic insufficiency.

Auscultation.—A loud harsh rough systolic murmur is heard over the base of the heart. It is usually prolonged and low pitched, but sometimes high pitched, and musical. The place of maximum intensity is the second right interspace. The murmur is also present at the level of the third rib over the sternum directly in front of the aortic orifice, and usually in the first and third interspaces to the right of the sternum. It is poorly conducted downward, but may be at times heard at the apex as a

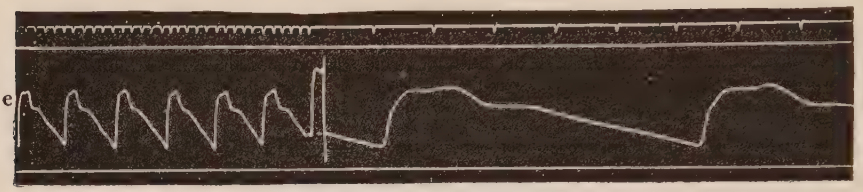


FIG. 113.—Pulse in aortic stenosis. Radial tracing made with the Frank-Petter sphygmograph. (Edens, "Lehrbuch der Perkussion und Auskultation," J. Springer.)

soft murmur. It is usually well transmitted to the suprasternal notch and the carotid arteries.

This murmur is one of the loudest that develop in valvular disease. Often it is distinctly audible over the back in the left interscapular region. Cases occur in which the murmur is present in more distant parts of the body. Stokes reported two cases in which it was heard over every superficial artery. One of his patients described his body as one humming top. The murmur follows the first sound after a slight but definite interval—the duration of the isometric period, which is .06 to .07 second. The

FIG. 114.—Radial pulse in aortic stenosis. Tracing made with the Frank mirror sphygmograph. Note the small size of the wave, the typical slow rise (pulsus tardus) and the well-marked anacrotic limb. (Romberg, "Krankheiten des Herzens und der Blutgefässe," Ferdinand Enke.)

murmur may be so loud as to be distinctly audible to the patient and heard by bystanders at a distance of three feet or more.

The second heart-sound at the apex and aortic area is weak or absent. When present it is usually the transmitted second pulmonic sound.

The Pulse.—In no other disease except aortic insufficiency is the form of the pulse wave so characteristic. It is a small long slow pulse wave

which raises the finger gradually. The upstroke in a graphic record is often interrupted by an anacrotic notch. The summit is rounded and the fall to the base line gradual. It is the *pulsus tardus*. The distinctive shape of the pulse wave is well shown in Fig. 113 made with the Frank-Petter sphymograph and in the tracing Fig. 114 obtained by means of optical registration. The pulse-rate is stated in all textbooks to be low. I know of no published statistics on the extent of retardation of the heart-rate or the percentage of cases in which it occurs. Osler says the rate does not often fall below 60, but occasionally it is constantly about 40.

The Blood-Pressure.—The systolic blood-pressure is low or of medium elevation and the diastolic pressure is increased; hence the pulse pressure is reduced.

Diagnosis.—The more skilled the physician the fewer are his cases of aortic stenosis in proportion to the total number of cardiac patients examined. The systolic murmur over the aortic area is of little aid in diagnosis because it occurs in so many other conditions. In fact very few cases which present systolic murmurs are affected with aortic stenosis. The murmur is present in the great majority of the cases of aortic insufficiency without stenosis. It is common especially in young people as a functional or inorganic murmur. In old subjects a systolic murmur is often due to roughening of the valves, atheroma or dilatation of the first portion of the aorta without stenosis. A loud systolic murmur is occasionally present in aneurism. Stenosis of the pulmonary orifice may be confused with aortic obstruction but the murmur is not transmitted to the vessels of the neck and is heard with maximum intensity to the left of the sternum. The subjects of this disease are young persons while as a rule aortic stenosis occurs in those of middle age. The average age in Broadbent's series was about forty, and the sclerotic form may be found in the aged. Osler refers to two cases that he had followed for years that lived beyond the age of ninety.

The presence of a systolic aortic murmur should simply serve to remind the examiner to feel the aortic area for a thrill and to note carefully the character of the pulse. To inexperienced observers any loud murmur at the base suggests stenosis whereas that lesion is the last to be considered.

The thrill is an important aid in diagnosis but may occur in other conditions. The association of the thrill with the typical murmur and the small slowly rising pulse occurs only in aortic stenosis.

The character of the pulse is distinctive. In deciding whether an aortic systolic murmur is due to an obstruction at the orifice the pulse is the criterion. If the pulse is sudden, large and collapsing we may conclude

there is no real stenosis. The extent of the lesion is not indicated by the loudness of the murmur, but by the shape of the pulse wave, and in some cases by the size of the heart.

The recognition of typical cases is easy from (1) the harsh loud systolic murmur over the aortic area, (2) the marked thrill that accompanies the murmur, (3) the slowly rising flat-topped pulse wave, (4) the associated aortic regurgitation, (5) the reduced pulse pressure.

Aortic Insufficiency

Deformity of the aortic valve leading to regurgitation of blood is a common form of valvular disease. It is possibly the most frequent, as mitral insufficiency of valvular origin, which statistics indicate to be by far the most common, is a diagnosis now known to have been often made when the disease did not exist.

Rheumatic infection is the most common cause of aortic insufficiency in some series of reported cases and syphilis in others. In Gerhardt's Basel clinic 57 of the 90 cases studied were of "inflammatory" origin, 14 due

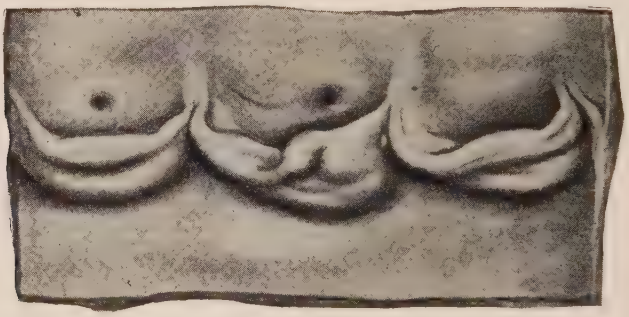


Fig. 115.—Flabby "wash-leather" thickening of aortic valve, which was notably incompetent. (Cowan, "Diseases of the Heart," Edward Arnold and Company.)

to lues and 9 to arteriosclerosis. In Romberg's Munich clinic on the other hand, 57 per cent were luetic, 17 per cent endocarditic and only 5.5 per cent arteriosclerotic. In the older statistical studies many cases that were really syphilitic were mistaken for the arteriosclerotic form. Syphilitic aortic insufficiency is often secondary to aortititis and rarely occurs before the fortieth year. A rare form of aortic insufficiency is due to septic endocarditis. The etiological agent may be the gonococcus, streptococcus viridans or other microörganisms.

Pathological Physiology.—With each diastole of the heart blood escapes from the aorta into the left ventricle. The amount that regurgi-

tates is difficult to estimate even in experiments on animals. It is probable according to MacCallum's studies that the backflow from the aorta is considerable. An incompetent aortic valve would, as Wiggers has shown, cause a marked drop in the diastolic arterial pressure even when little blood flowed back into the ventricle. The essential dynamic disturbance —the collapsing pulse—is brought about not by the volume of blood which regurgitates but by the fall of pressure during diastole. Hewlett demonstrated by volume curves that in aortic insufficiency there was a definite flow of blood from the smaller to the larger vessels of the arm during diastole, and Duroziez's sign indicates a backward movement of blood in the femoral artery during this portion of the cardiac cycle. If the amount of regurgitated blood is considerable and the inflow from the left auricle not decreased the ventricle will be dilated with the blood at the end of diastole. This distention of the ventricle increases the initial tension and the heart muscle is able to contract with greater force during the following systole. For additional discussion of the dynamics in aortic insufficiency, see pages 264, 267. The left ventricle undergoes marked hypertrophy. If the heart muscle weakens congestive dilatation occurs. In experimental aortic insufficiency there is no congestion of blood in the left auricle or in the pulmonary vessels. In the cases seen clinically as long as the extra work caused by the leaky valve is fully done by the left ventricle there is no dilatation of the left auricle or chronic passive congestion of the lungs. After the left ventricle fails the blood is dammed back in the left auricle and the pulmonary veins. More work is thrown on the right ventricle when the flow of blood in the lungs is impeded and it becomes hypertrophied. Later the right ventricle becomes insufficient and dilatation of the cavity and the right auricle occurs. This is followed by congestion of the abdominal organs and subcutaneous edema.

Inspection.—Aortic insufficiency is the only valvular disease in which the diagnosis can be made at sight. Such throbbing of the peripheral arteries as is present in advanced aortic insufficiency occurs in no other disease. This is frequent in cases of rheumatic origin as they occur at an age when the elasticity of the vessels is not impaired and is much less common in the luetic and sclerotic forms of the disease. The visible throbbing of the carotids, along their entire course, together with the pulsation in the temporals and radials suggests the diagnosis. The head even may jerk with each heart beat, and on careful observation it will be seen that the head is thrown back at each systole (Musset's sign). The vessels suddenly expand during systole and then quickly empty. There is often in severe cases a striking pallor of the face (aortic facies). This is ascribed

to the small amount of blood furnished to the capillaries during the greater part of diastole.

There may be such a marked pulsation in the sternal notch or in the second right interspace as to suggest aneurism. The cardiac impulse is visible as an outward thrust in the region of the apex and is usually seen only over a small area, if the heart muscle is strong. After the onset of congestive dilatation, the impulse is diffuse and widespread, and it may extend outward to the anterior or even to the mid-axillary line and downward to the seventh or eighth interspace. If the heart is very large and strong there may be a definite systolic heave of the entire left front of the chest. In children and young persons the yielding thorax will bulge in the cardiac region, if there has been marked enlargement of the heart for some time. The *capillary pulse* is seen in most cases (page 359). Occasionally a remarkable form is present. When the hands are held up against the light the palms blush in systole and turn pale in diastole. This change in color has been visible six or eight feet away. (Osler.) The *progressive venous pulse* (page 361) is sometimes present particularly in the veins of the back of the hand. To bring out this sign, the hand should be dropped at the wrist so that the veins will fill. The pulsation if present is "a gentle and deliberate movement followed with difficulty" (Broadbent.)

Palpation.—The cardiac impulse is characteristic of hypertrophy of the left ventricle. A heaving thrust develops early in the disease, but it is palpable only over a small area. The position of the apex impulse is dependent on the amount of dilatation. It is usually dislocated to the left and downward. With increase of dilatation, the palpable impulse spreads over a wider area. The strength of the impulse is not a measure of the strength of the heart (Martius). This is a point that must be emphasized. A powerful cardiac impulse may result largely from a congestive dilatation and with an improvement of the condition of the cardiac muscle the force of the impulse may actually lessen. Neither the force nor the form of the impulse enables one to distinguish between hypertrophy or dilatation. In cases of rheumatic aortic insufficiency in young persons the impulse is as a rule stronger than in the luetic or sclerotic forms in older persons and this is independent of the degree of hypertrophy or dilatation. Occasionally a diastolic thrill is present at the aortic area.

Percussion.—The cardiac dullness is usually increased to the left and downward. It is rare to have it spread in an upward direction. If congestion dilatation is absent the percussion outline may be normal. The enlarged area of cardiac dullness often has an elongated form. The left border of dullness may be outside the apex impulse. Occasionally great

dilatation of the left ventricle pushes the right heart to the right so that its border assumes the position it occupies in dilatation of the right auricle.

There is frequently in cases of long duration dullness over the manubrium or at the sternal end of the second right interspace. This is due to dilatation of the aorta. For the form and size of the heart as revealed by radioscopic examination see page 399.

Auscultation.—Very important in diagnosis is the diastolic murmur that develops at the aortic orifice and is heard at the base of the heart. The character and the location of the murmur differ in individual cases. The murmur is sometimes loud and blowing. Cases do occur, although they are rare, in which the murmur is audible at some distance from the patient. These distance murmurs, as they are termed are much less frequent in aortic insufficiency than in aortic stenosis (v. Jagić). On the other hand, the murmur may be so soft as to be heard with difficulty. Its quality may be rough; rarely it is musical. The point of maximum intensity may be in the second right interspace but more often it is in the third left interspace or over the third left rib. Occasionally the murmur is loudest over the middle of the sternum. It is well transmitted downward along the left border of the sternum and sometimes it is heard more distinctly in the left axilla than in the usual location. (Cole.) It may be conveyed to the apex and is occasionally heard loudest here. (Mackenzie). It is questionable if the murmur is ever confined to the apex region as has been claimed. Unlike the systolic murmur it is rarely well transmitted to the vessels of the neck. The murmur may be louder over the pulmonary than over the aortic area. This may lead beginners into the error of regarding the murmur as due to pulmonary insufficiency. The pulse and other physical signs indicate its true place of origin, when it is best heard over the pulmonary area. The murmur of aortic insufficiency begins early in diastole with the second sound or in place of the second sound and rapidly diminishes in loudness. The murmur may vary in its duration as in its other characteristics. Although it may be a short whiff, it is usually long, drawn out, and may occupy the entire diastole. If the murmur is soft and faint, it may sometimes be heard better with the ear applied directly to the chest than with the stethoscope (Flint). The murmur may be louder in the recumbent position than in the erect posture, or only audible in the recumbent position. On the other hand it may be heard when the patient is sitting up and disappear when he lies down (Romberg). Pardee found it was most distinct when the patient stood and bent forward, and that it was intensified by exercise.

The Second Aortic Sound.—It may be loud and ringing but more often is enfeebled or absent. If aortic stenosis exists the second

sound will not be heard. Osler held that in the majority of cases of aortic insufficiency, the second sound disappeared.

There is usually a systolic murmur over the aortic area. It was absent only eighteen times in D. Gerhardt's series of ninety-nine cases. Only rarely is it caused by a stenosis. It may arise simply from the abnormally large and rapid output of blood from the ventricle. A systolic murmur at the apex often results from mitral insufficiency which may be due to mitral endocarditis but more commonly results from a dilatation of the mitral ring of muscular tissue (muscular insufficiency).

Flint Murmur.—According to most American writers a rumbling decrescendo murmur at the apex region occurring in the middle or latter part of diastole is common in aortic insufficiency. This was first described by Austin Flint. Thayer's statistics showed it was found in seventeen out of forty-six cases of pure aortic insufficiency studied at the Johns Hopkins Hospital. Osler taught that it was of common occurrence. This opinion is not shared by all authorities. Gibson of Edinburgh regarded it as very rare. He was on the watch for the Flint murmur and saw cases frequently, he says, in which with absolute evidence of aortic disease there was a presystolic murmur, but in every one of these post-mortem examination revealed mitral obstruction as well as aortic lesion. D. Gerhardt observed it in only three cases, and Romberg writing as late as 1921, asserts that he has never heard it. White in a recent paper raises the question of the frequency of the Flint murmur. He believes that "a mitral diastolic murmur in a rheumatic heart should generally be considered as a combination of aortic regurgitation and organic mitral stenosis, in a syphilitic heart as aortic regurgitation and functional mitral stenosis." Flint stated that the murmur is not constant, but is now present and now absent and that it is limited to a circumscribed space around the apex. The murmur, Flint held, was strictly presystolic in time, for he says "at the time when the auricular contraction takes place, the left ventricle is already filled with blood, the mitral curtains are floated out so as to be in contact with each other, and the mitral direct current passing between the curtains throws them into vibration precisely as when the orifice is narrowed."

The diagnosis of a Flint murmur should not be made without careful consideration of every detail of the case. There is no doubt of its existence. The combined clinical and post-mortem observations of Flint, Osler and other able physicians prove this without a shadow of doubt. I believe, however, that it is of rare occurrence. If the murmur is mid-diastolic it certainly does not correspond to Flint's description, and the probability is strong that it is either due to mitral obstruction or is the

aortic murmur itself modified in character in its transmission to the apex.

The Pulse.—A large pulse wave suddenly striking the finger and leaving the artery apparently empty between the beats is characteristic of aortic regurgitation. It is called the water-hammer or collapsing pulse; sometimes the Corrigan pulse after its discoverer. Hope called it the pulse of unfilled arteries. The most typical feature of the pulse is the sudden violent rush of the wave which fills the vessel and lifts the fingers forcibly. It is short as well as sudden and the artery may seem to collapse beneath the palpating finger. The collapse, however, may be more apparent than real (Wiggers). That its occurrence may be demonstrated by accurate methods is shown in Fig. 116. This tracing was made

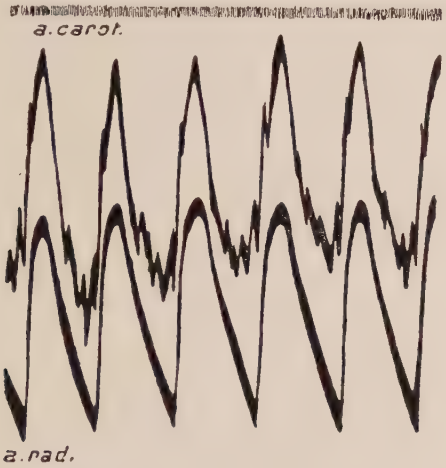


Fig. 116.—Pulsus celer with a variation of blood-pressure from a maximum of 200 to a minimum of 40 mm. Hg. in aortic insufficiency. Tracing of the carotid and radial pulse with Frank's mirror sphygmograph. Time above in 1/50 second. Note that the downstroke is almost as sharp as the upstroke. This is a true collapsing pulse. (Romberg, "Krankheiten des Herzens und der Blutgefässe," Ferdinand Enke.)

with Frank's optical sphygmograph and the descent of the pulse is seen to be almost as sudden and sharp as the ascent. The wrist should be held above the shoulder in order to bring out the characteristic features of the pulse.

The heart-rate when the heart muscle is strong is more rapid as a rule than in the other valvular lesions during the stage of so-called compensation. In this disease the heart and blood vessels are particularly susceptible to mental stimuli. Slight emotional disturbances elevate the heart rate and produce changes in the calibre of the peripheral vessels (Mackenzie).

The arteries are wide, elongated and frequently tortuous. Thickening

of the vessels from sclerotic changes occurs frequently even in young persons.

Arterial Sounds.—A loud sudden explosive sound occurring during the cardiac systole may be heard over the larger arteries, especially the femoral and brachial arteries. This is called the pistol shot sound. A double sound is often audible over the femoral artery (Traube's double sound). This is due to the sudden tension and subsequent relaxation of the arterial walls. Occasionally in debilitated patients with rigid arteries only a diastolic sound or a split diastolic sound is present (Gerhardt). Very rarely three sounds are audible over the femoral artery.

A double murmur over the femoral artery is of some diagnostic importance in aortic insufficiency (Duroziez's Sign). Occurring spontaneously without any pressure of the stethoscope this phenomenon is rare and is heard only in aortic insufficiency (v. Jagić). Pressure of a certain degree which varies in different cases often brings out this murmur in this disease. A loud systolic murmur precedes the softer diastolic murmur. The diastolic portion becomes louder by compression of the artery below the point ausculted. This pressure murmur may occur in other conditions than aortic insufficiency when the tone of the vessel walls is relaxed. The arterial sounds are produced locally in the artery by the impact of the pulse wave on the blood column (Jamieson and Wilson).

The relative frequency of the various arterial signs in aortic insufficiency has been determined by Tice in a series of 124 cases. The Corrigan pulse was present in 95 per cent of the cases, the capillary pulse in 90 per cent, Duroziez's murmur in 88 per cent, visible arterial pulsation in 76 per cent, a pistol shot sound in 45 per cent, Traube's double sound in 24 per cent, and a visible venous pulse in 7 per cent.

Blood-Pressure.—The diastolic pressure is abnormally low, unless aortic stenosis is present. The pulse pressure is greatly increased. It is often 100 mm. of mercury or more instead of the normal 30 or 40 mm. The systolic pressure is usually elevated and is frequently between 150–170. The fourth phase must always be taken as a measure of the diastolic pressure by the auscultatory method as some sound is frequently present in the brachial artery when no pressure is applied. The systolic blood-pressure is much higher in the femoral artery than in the brachial. White refers to a case in which the systolic pressure in the brachial was 136 mm. and in the femoral 270 mm. Higher pressure in the femoral than in the brachial artery also occurs in primary hypertension and after muscular exercise in normal persons. This increased pressure in the leg may be due to increased arterial tonus, maintained as a safety device to provide adequate blood supply to the brain.

Diagnosis.—During the World War this valvular lesion was the one most frequently overlooked. In cases of marked regurgitation the vascular signs make the diagnosis easy. In fact Corrigan in his classical paper makes no mention of the diastolic murmur. This was first described by Hodgkins.

In cases of slight regurgitation the diagnosis must often be largely based on the presence of a soft blowing murmur beginning early in diastole and heard over the sternum or along the left sternal border. Additional evidence is often furnished in mild cases, in the absence of arterial signs, by a heaving apex impulse, enlargement of the outline on percussion, the characteristic shape of the orthodiagram, and an electrocardiogram indicating left sided preponderance.

The murmur may be absent in advanced aortic insufficiency. The diagnosis can still be made by the characteristic pulse signs and the size and shape of the heart.

The altered diastolic murmur in mitral stenosis after the onset of auricular fibrillation is frequently mistaken for an aortic murmur. Auricular fibrillation in aortic disease is rare.

Functional aortic insufficiency may occur but very few cases have been reported and *only in pernicious anemia.* The diastolic portion of a venous hum and a diastolic murmur of cardiorespiratory origin have been mistaken for the murmur of aortic regurgitation.

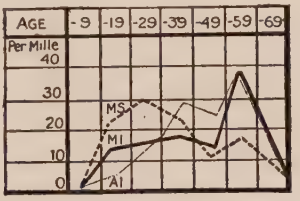


FIG. 117.—Diagram showing relative frequency of the most important valvular lesions at various ages. (Modified from Gillespie.) Solid line, mitral insufficiency; broken line, mitral stenosis, dotted line, aortic insufficiency. The figures indicate the decades. Under 9 years, –9; 10–19, –19; 20–29, –29; etc. (Hirschfelder, "Diseases of the Heart and Aorta," J. B. Lippincott Company.)

Mitral Stenosis

Occurrence.—Mitral disease occurs about twice as frequently as aortic disease. This is shown by an analysis of 1781 cases of valvular heart disease, admitted to the Johns Hopkins Hospital (Hirschfelder) and confirmed by the autopsy statistics collected by Sperling in Berlin.

Mitral stenosis is the most common form of valvular heart disease.

It is usually combined with mitral insufficiency. In cases of severe cardiac weakness in which the physical signs of mitral regurgitation are marked, there is generally a stenosis also present, although its recognition may be difficult.

In mitral stenosis of the pure type, that is without demonstrable insufficiency, the narrowing of the orifice is of high degree. Mitral stenosis with the opening reduced to a small calibre can exist for years without the production of a murmur or any evidence of cardiac enlargement. I have studied clinically and pathologically one such case. The patient had recurring attacks of tachycardia, probably due to auricular flutter, and during a period of nearly ten years she was examined by a number of physicians including the late Dr. Tyson of Philadelphia, an able diagnostician, and the existence of mitral disease was never suspected. I examined her repeatedly and never heard any murmur. The orthodiagram was of normal size. Death occurred quite suddenly during an attack of tachycardia. The autopsy revealed a mitral orifice of the buttonhole type and also tricuspid stenosis. Romberg states that he has seen two similar cases. They are of course very rare, but their occurrence proves that great stenosis can exist without producing physical signs.

Etiology.—A history of rheumatic fever of chorea is obtained in fifty or more per cent of the cases of mitral stenosis. In Gerhardt's carefully studied series of 155 cases of mitral stenosis there was a history of rheumatic fever in 107 or 69 per cent. Rheumatic infection is the only disease that is definitely known to cause mitral stenosis. It is possible that arteriosclerosis may cause this valvular lesion, but in Gerhardt's large series only four were attributed to this disease. There is a large number of cases in which no history of any infectious disease can be obtained. The French have regarded these cases, many of which are pure mitral stenosis, as the result of tuberculosis, but with little evidence to support the theory. Chlorosis is also regarded as a cause. Personally, I believe that rheumatic infection is the cause of all or nearly all of these cases of mitral stenosis. The fact that no history of rheumatic fever is obtained is no argument against this conclusion for the reason that the symptoms of rheumatic infection in the joints and muscles may be so slight that no attention is paid to them. I treated for many weeks, a little girl with severe rheumatic endomyocarditis in whom the only symptom of rheumatic arthritis was slight pain with weakness in one hand lasting only one or two days. In regions where rheumatic infection is usually a progressive one and the orifice is frequently, in advanced Atlanta, Birmingham and New Orleans, mitral disease is unusual, and I

have found on personal inquiry that rheumatic fever and chorea are far less common than in the North. If nearly half of the cases of mitral stenosis are due to tuberculosis and infectious diseases, other than rheumatism, such as measles, scarlet fever, and whooping cough which are prevalent in the South, then one would expect to find about half as many cases of mitral stenosis in the southern clinics as in regions where rheumatic infection is common, but the number is far less. Hence the conclusion seems reasonable that mitral stenosis is in the vast majority of cases, the sequel of a previous rheumatic infection.

Pathological Anatomy.—The mitral valve is thickened and deformed, and often the two segments are fused together. (Fig. 118.) The condi-

Fig. 118.—Stenosis of the mitral. The valve is considerably thickened as is shown in the cross-section *a* and *a*. The chordae tendinae are thickened and shortened. The circumference of the valve measures from *a* to *a* 4.5 cm. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," F. C. W. Vogel.)

tion is usually a progressive one and the orifice is frequently, in advanced cases, reduced to a mere slit, "the buttonhole contraction," or to a round hole only a few millimeters in diameter at the bottom of a funnel. The valves may be calcareous or very hard and dense from an overgrowth of connective tissue. The chordae tendinae are thick and shortened. The process is not limited to the valves but the myocardium is always involved to a greater or less extent. The papillary muscles are partly converted into connective tissue. The auriculoventricular bundle lies near the mitral valve and is frequently the seat of chronic inflammatory changes. As a result heart-block is more often associated with mitral disease than with other valvular lesions. The involvement of the auricular muscle in some way probably causes auricular fibrillation which is a very frequent disturbance of the heart rhythm in mitral stenosis.

Pathological Physiology.—Narrowing of the mitral orifice throws additional work on the left auricle. The lumen can be reduced in diameter about two-thirds before any considerable increase in the work of the heart is necessary. With increasing stenosis the burden thrown on

the left auricle rapidly increases (Lewy). The auricle undergoes hypertrophy and dilates, and sometimes reaches an enormous size. In one case its capacity was increased from the normal 60 cc. to 3000 cc. (Minkowski). The blood becomes dammed back in the pulmonary veins. Later the pressure rises in the pulmonary artery as a result of interference with the onflow of blood through the lungs. The right ventricle is then called upon to do more work and becomes hypertrophied. When the burden becomes too great it undergoes congestive dilatation. The right auricle then dilates, the tricuspid valve becomes insufficient, and back pressure is exerted on the blood in the jugular veins, the liver, and kidneys causing chronic passive congestion.

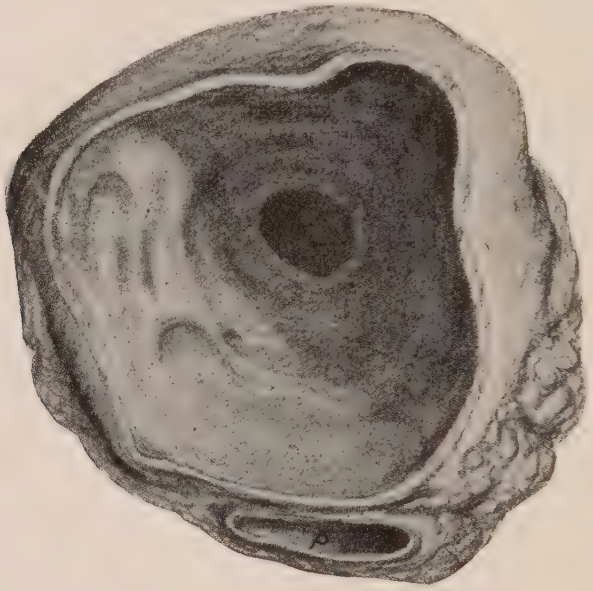


FIG. 119.—Stenosis of the mitral. The greatly dilated auricle has been opened and is seen from above. In the bottom of the cavity is the narrowed round orifice of the mitral. *P.* pulmonary artery. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," F. C. W. Vogel.)

Normally, Henderson holds, the filling of the ventricle takes place rapidly after the opening of the auriculoventricular valves. Only a small volume is added by the auricular contraction. The blood flow from the left auricle to the left ventricle in experimental mitral stenosis has been studied by Hirschfelder. The effect of mitral stenosis of moderate degree is the filling of the left ventricle by the left auricle more slowly than normal during the early part of diastole. The left auricle contains as a result of the mitral obstruction more blood at the onset of its contraction and forces a larger amount of blood into the

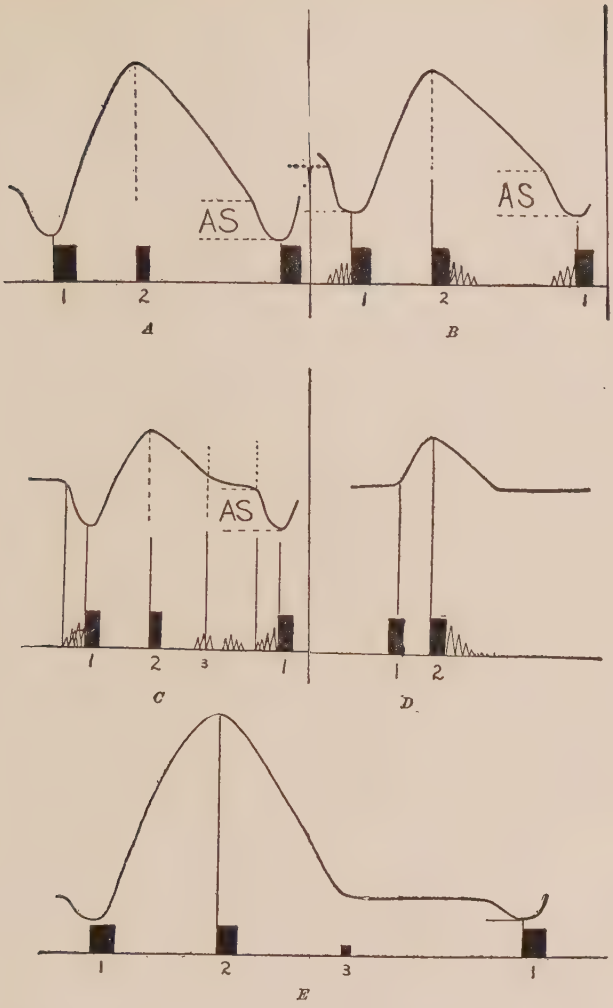


FIG. 120.—Diagram of volume curve of ventricles of the dog's heart in progressively increasing experimental mitral stenosis. The recording tambour of the cardiometer was inverted. The upstroke corresponds to the emptying of blood from the ventricle into the aorta during systole. This is followed by the immediate filling of the ventricles, represented by the descent of the curve. The dotted verticle line marks the beginning of diastole. A. Normal curve at normal rate. AS = portion of filling due to auricular systole. B. Mitral stenosis, mild grade, showing decreased volume, slowed filling, and greater importance of the auricular element in the curve. C. Medium grade of stenosis, showing diminished filling, early onset of diastasis, and the large amount of output from the auricle. D. Severe grade of mitral stenosis (third stage), small systolic output, early diastasis, absence of auricular systole. E. Normal volume curve, slow heart-rate, showing diastasis under normal conditions. Solid black lines represent normal heart-sounds, zig-zag lines represent murmurs. (A. D. Hirschfelder, Johns Hopkins Hosp. Bull., Balt., XIX.)

ventricle, Fig. 120. The upstroke in this diagram represents decrease and the downstroke increase of volume. With progressive stenosis there is a diminished filling of the left ventricle; the systolic output lessens and the blood-pressure falls unless it is maintained by constriction of the peripheral arteries as a result of vasomotor action. The experimental method used by Hirschfelder has been criticised by Wiggers, and H. Straub on the basis of recent work maintains that the auricular systole normally contributes one-third to two-thirds of the whole ventricular contents. This is directly opposed to the earlier work of Henderson. Clinical observations, however, support the view that the auricle is filled in great part without the aid of auricular systole. In many cases of mitral stenosis with auricular fibrillation, which is virtual paralysis of the auricle, the pulse is of good volume, no congestion is present in the lungs and considerable demands can be made on the heart in the form of exercise without discomfort. In one of my patients with cardiosclerosis, who was well digitalized, not only did no breathlessness or other symptoms develop with the onset of auricular fibrillation, but the vital capacity, which is an indirect method of determining the reserve power of the heart, even rose for a time. This certainly indicated that the heart was well supplied with blood and that it was not dammed back in the pulmonary veins.

In pure mitral stenosis there is no hypertrophy of the left ventricle, and actual atrophy may occur. Usually in the ordinary form of mitral stenosis there is some hypertrophy and dilatation of the left ventricle due to the associated mitral insufficiency. In a series of thirty-two cases examined at autopsy by Sansom, the left ventricle was normal in twelve, enlarged in seventeen and small in three. Out of sixteen cases in which the separate ventricles were weighed the left was smaller than normal in six (Lewis).

Inspection.—The face has often a ruddy hue and the lips are of deeper color than in health. There may, however, be pallor. After the onset of cardiac weakness the jugular veins are apt to be engorged and their pulsations often prominent. The apex impulse may be in the normal position but is usually displaced to the left. It is sometimes found no lower than the fifth interspace even in advanced disease, but may be in the sixth or seventh. The visible pulsation often extends over the fourth and fifth left interspaces from the sternal border to beyond the parasternal line. There is frequently epigastric pulsation. This is usually attributed to the hypertrophy of the right ventricle, but Lewis has shown that the extent of this impulse bears no relation to the weight of the right ventricle. Sometimes a visible pulsation is seen to the right of the sternum.

Palpation.—The apex impulse is increased in force. It usually gives

a sharp tap to the fingers entirely different from the forceful heave of the heart in rheumatic aortic insufficiency. Pardee calls the typical impulse in mitral disease sharp and slapping.

The typical presystolic thrill is a valuable sign in diagnosis of mitral stenosis. Its significance is so great that in some cases the diagnosis may be made by palpation alone. The examiner must disregard mere quivering impulses and slight vibrations and limit the term to well-marked vibrations like that of a purring cat. It was Laennec who invented the name "frémissement cataire" for this physical sign. As Gee truly says what may be considered a well-marked vibration can be learned from experience only. The true thrill of mitral stenosis is limited to the apex region and ends in the shock of the first sound. It is occasionally felt only after exercise and is most marked in expiration. "The localization, the occurrence in diastole, the purring vibratory quality, and the abrupt termination in the first sound, form a quartet of signs that rarely lead us astray." (Osler and Gibson.) A slight presystolic thrill in the early stages of the disease may precede the murmur by several years according to Mackenzie. One would not be justified in making the diagnosis on a slight thrill in the absence of the murmur. There is no doubt, however, that a true thrill alone may be present. If the vibrations are below 16 per second they can be felt but are too low pitched to be audible, and on the other hand if the number of vibrations exceeds 640 to the second no thrill will be produced no matter how loud the murmur may be. (Leichtenstern.)

Percussion.—The relative and absolute cardiac dullness is increased to the left and if in addition to the stenosis the mitral insufficiency is considerable the dullness may extend downwards to the sixth or seventh interspace. The upper border of absolute dullness may be as high as the third left rib if the pulmonary artery and left auricle are dilated. The increase of dullness to the left in mitral stenosis results from enlargement of the right ventricle. Contrary to the old view there is rarely any definite increase in the dullness to the right of the sternum until dilatation of the right auricle has occurred. Dilatation of this chamber is indicated not only by displacement of the right border to the right but more definitely in many cases by the distension of the jugular veins.

Orthodiagrams and teleradiograms show a heart of characteristic shape in mitral disease. The second arc on the left border is prominent due to the enlarged left auricle and the border of the left ventricle forming the third arc is of normal contour. The right border formed by the right auricle bulges and is markedly convex if the auricle is dilated. For additional details on the orthodiagram in mitral disease, see page 400.

Auscultation.—There is at the apex a diastolic murmur which is the most important sign in the diagnosis of this disease. The murmur may occur toward the end of diastole, the so-called presystolic murmur, or it may begin in early diastole and end in mid-diastole, or extend throughout the entire diastole.

The Presystolic Murmur.—It is one of the most striking auscultatory phenomenon which the student meets in the study of physical diagnosis. Yet it was not discovered until physicians had been listening to cardiac murmurs with the stethoscope for more than twenty years. M. Fauvel in 1843 described it as a loud rough murmur, running up to and terminating with the first sound. He recognized that it was pathognomonic of mitral stenosis and gave it the name of presystolic murmur. The better name of auriculosystolic murmur given to it in 1861 by Gairdner of Glasgow has never come into general use. The murmur is heard only over a limited region, even when very loud. Balfour demonstrated in his clinical lectures the limitation of the propagation of this murmur most strikingly. In one of his patients the murmur was easily recognized through three shirts, two of them flannel, a waistcoat, coat and overcoat. Yet when stripped this murmur was not distinctly audible above the third rib or below the sixth interspace. In some cases the area over which the murmur may be heard can be covered with the bell of the stethoscope. It is crescendo in character and ends in the shock of the first sound. The murmur may be phonetically represented by the symbols *rrrb* or *voot* when vocalized (Balfour). As the second sound may be weak or absent and the first sound valvular in character, beginners often mistake the presystolic murmur for a systolic murmur replacing the first sound. This can be avoided by noting that the murmur precedes the apex impulse and the carotid pulse. If the heart-rate is rapid it will be impossible to time the murmur accurately. With the onset of auricular fibrillation this murmur disappears, although it may be simulated by the diastolic murmur. This resemblance occurs during the shorter cardiac cycles when no interval of silence separates the diastolic murmur from the first sound. The first sound in which the murmur seems to end then gives it in some way a more or less crescendo quality. During the longer diastolic pauses in auricular fibrillation it is easy to perceive that this same murmur has really a decrescendo character and dies away in silence before the first sound is heard.

If heart-block is present in mitral stenosis the crescendo presystolic murmur may be separated from the first sound by a brief interval of silence due to the delay between the auricular and ventricular systole. This definite separation from the first sound furnishes additional evidence

not only of the existence of heart-block, but also, as Cohn [1] first pointed out, that it is the auricular systole which causes the presystolic murmur. A number of investigators during the past twenty years, including Brockbank, Sahli, and Henderson have claimed that this murmur is really systolic in time and due to the ventricular contraction.

The Diastolic Murmur.—This begins a brief interval after the second heart-sound and continues for a varying length of time. It may end in mid-diastole or continue nearly to the end of diastole. This murmur is positive evidence of mitral obstruction. In a sense it is more characteristic of the disease than the presystolic murmur as, unlike the latter, it persists after the development of auricular fibrillation. This shows the "greater need of relying on the diastolic rather than on the presystolic murmur in the diagnosis of stenosis" (White). The murmur is usually in early cases faint and of short duration but as the obstruction increases it becomes louder and longer and may then fill the whole diastolic period. It is of diminuendo character. When faint it is described as a hum, but when louder it is usually a low-pitched rumble. It is heard over a limited area at or just inside the apex impulse. An appreciable period of silence separates the murmur from the second sound. This distinguishes it from the murmur of aortic insufficiency which begins with or immediately after the second sound. Frequently when the heart-rate is slow the long diastolic pause is nearly filled with a murmur which begins loudly in early diastole, falls away in mid-diastole, and ends loudly in presystole. This falling and rising murmur is due to the fusion of the diastolic and presystolic murmur.

The diastolic murmur is due to the rapid gush of blood, that has accumulated in the auricle, through the narrowed mitral orifice when the valve opens early in diastole.

Effect of Position and Exercise on Diastolic and Presystolic Murmurs.—These murmurs are best heard with the patient recumbent and lying on the left side. They are sometimes loud in this position when inaudible in the erect posture. Exercise or the inhalation of amyl nitrite increases the intensity of the murmurs. Examination for a mitral diastolic murmur is never thorough until the effect of posture and exercise has been carefully observed. As the murmur may quickly disappear no time should be lost after the exercise is finished in listening for the murmur in the recumbent position.

The early diastolic murmur has usually been regarded as developing much later in the course of the disease than the presystolic murmur but White, a most trustworthy clinical observer, asserts that he has rarely

[1] *Arch. Int. Med., Chicago,* 1915, XV, 758.

heard a pronounced presystolic murmur in the absence of the mid-diastolic murmur.

Heart-Sounds.—The *first sound at the apex* is usually sharp and loud. It often has a valvular ringing quality that resembles an accentuated aortic second sound. Rarely it may be so loud as to be heard several feet from the patient. In one of Osler's cases it was audible at a distance of six feet. The loud first sound is probably due to the small volume of blood in the ventricle at the onset of its contraction and the resulting low intraventricular pressure. (Robinson.) *The second sound at the apex* may disappear in advanced cases especially when there is severe cardiac weakness. The second pulmonic sound is usually markedly accentuated. This is caused by the high pressure in the pulmonary artery.

The Third Heart-Sound.—This is frequently heard in mitral stenosis. It is sometimes present in the absence of the diastolic murmur, but when the diastolic murmur is audible, it follows immediately the third sound. The *punctum maximum* is usually in the region of the apex, but may be near the sternum in the fourth interspace and more rarely at the base. Its intensity varies from a faint sound to one that equals or exceeds that of the other two heart-sounds (Gerhardt). Its mode of origin is not clear.

Graham Steell Murmur.—This is a diastolic murmur heard best in the second left interspace, and ascribed to functional pulmonary insufficiency resulting from dilatation of the orifice of the pulmonary artery. Its discoverer, Graham Steell, found it rarely, but some American clinicians claim that it is present in about one-third of the well advanced cases of mitral stenosis. I agree with White that one should be very cautious in calling an early diastolic murmur present at the left sternal border a Graham Steell murmur. Most of the cases in which it was present have been found at autopsy to have aortic insufficiency. There is no doubt pulmonary regurgitation is more often diagnosed than existent.

The Pulse.—There is no characteristic change in the pulse while the circulation is well maintained. Auricular fibrillation develops in a large proportion of the advanced cases and then the pulse becomes absolutely irregular. When heart failure develops the pulse wave usually becomes small and the systolic blood-pressure may be low.

The Electrocardiogram.—There is often evidence of right ventricular preponderance. The auricular complexes (*P* waves) in Lead II are prominent. Their duration is often lengthened and the summit frequently notched. The *P–R* interval may be increased, due to delay in the conduction of the impulse from the auricle to the ventricle (heart-block).

Diagnosis.—The experience of the war showed that many cases of rapid forcible heart action due to nervousness were mistaken for mitral stenosis. In these cases a reduplicated heart-sound was frequently mistaken for a presystolic murmur, and a forceful quivering impulse for a thrill. When a typical thrill and presystolic murmur are present no valve lesion is more easily recognized. If the heart action is rapid and the observer has not learned from experience these distinctive signs he should not base a diagnosis of this serious disease on a presystolic thrill and murmur. The *diastolic murmur* is a more trustworthy sign as White has pointed out. In cases of pericardial adhesion a rumbling apical murmur may be present but it rarely is so limited in area nor does it end in a snapping first sound (Osler and Gibson).

In cases of well-marked mitral insufficiency with symptoms of cardiac failure and in all cases of auricular fibrillation in persons under thirty-five years of age the existence of mitral stenosis is probable even if no diastolic murmur is present.

Mitral Insufficiency

Mitral regurgitation may be due to valvular endocarditis or to dilatation of the ring of muscular tissue around the mitral orifice. It occurs in nearly all cases of mitral stenosis.

The valvular form is usually the result of rheumatic infection. It may also result from septic endocarditis. Mitral regurgitation of muscular origin due to dilatation of the mitral ring frequently occurs in cardiosclerosis. In mitral endocarditis there is usually involvement of the muscular ring as well as of the valve. In the majority of cases "the change in the valves is altogether inadequate to explain the evidently free regurgitation that occurred during life, and the diastrous dilatation of the heart. The muscle-failure factor, it may be presumed, was the essential one." (Steell.)

Pathological Physiology.—For the cardiac dynamics in mitral insufficiency, see page 268. After injuring the mitral valve the carotid pressure falls and the mean left auricular pressure rises. The pressure in the left auricle in early ventricular systole rises markedly instead of falling rapidly as it does normally. Experimental work has not supported the theory that a mitral regurgitation directly causes a damming back of blood in the pulmonary artery. The pressure in the pulmonary artery in experimental lesions is altered but little. (MacCallum and McClure.) As long as the left ventricle is efficient, the

great capacity of the auricle and the pulmonary veins when distended enables them to accommodate the blood ejected into the auricle by the left ventricle. In clinical cases when the left ventricle begins to fail, the lungs become congested and the pressure in the pulmonary artery rises. There is hypertrophy and dilatation of the left ventricle and left auricle and later hypertrophy of the right ventricle. Dilatation of the right ventricle when it occurs, is of the congestive type, and due to weakness of its muscle.

The presence of fibrous nodules, rough edges and vegetations on the mitral segments does not necessarily result in insufficiency as smooth rods and even knotted strings have been introduced experimentally and the valves by their elasticity, aided by the sphincter-like action of the muscular ring surrounding the orifice, prevented regurgitation. (de Sautelle and Grey.)

Physical Signs.—There may be a visible pulsation over the entire cardiac region especially prominent in many cases between the apex region and the xiphoid cartilage. In well-marked mitral regurgitation a systolic thrill may be present at the apex but it is rare. The cardiac impulse is heaving and displaced outward and sometimes downward if there is well-marked hypertrophy and dilatation of the left ventricle. The cardiac dullness is increased chiefly to the left and above. An increase to the right of the sternum only occurs when dilatation of the right auricle has taken place as a result of weakening of the right ventricle. The Roentgen examination shows a bulging of the second left arc due to dilatation of the left auricle. This may obliterate the normal angle in the left border of the cardiac shadow that marks the junction of left auricle and ventricle. See section on orthodiagram in mitral disease page 400.

The murmur in early cases of rheumatic mitral insufficiency is often soft and blowing. In old cases it is usually loud and harsh and sometimes has a musical quality. It may obscure or replace the first heart-sound. The point of maximum intensity is the apex but it may be occasionally equally loud over the pulmonary area in the second left intercostal space. The second pulmonary sound is accentuated and may at times produce a palpable shock.

Diagnosis.—Usually considered the easiest valvular lesion to recognize it is frequently the most difficult. Even to-day the diagnosis is often based entirely on the presence of a systolic murmur at the apical region, although it is common knowledge among well-trained physicians that most systolic murmurs are not due to mitral insufficiency of valvular origin. Loud harsh murmurs especially if accompanied by a thrill are

almost always due to disease of the valve but occasionally may be functional. The murmur is frequently transmitted to the axilla.

The diagnosis is not justified unless two of the three following criteria are present (Rothschild): (1) A systolic murmur at the mitral area; (2) a heaving apex impulse (hypertrophy) or an apex impulse displaced outward beyond the mid-clavicular line (dilatation). This finding should be supported by a definite enlargement of the areas of absolute and relative cardiac dullness; or better an orthodiagram showing the characteristic shape of the heart in mitral disease and with the diameters increased; (3) a history of rheumatic fever.

The discovery of the diastolic murmur of mitral stenosis may be regarded as evidence that a systolic murmur indicates the existence of insufficiency of valvular origin. In all cases of mitral insufficiency repeated examination should be made at regular intervals for the murmur of mitral stenosis. The heart, the seat of mitral disease of rheumatic origin, at first develops insufficiency and later stenosis.

In persons over forty years of age presenting evidence of cardiac enlargement, but without a history of rheumatic infection a systolic murmur is to be regarded as probably due to muscular insufficiency. This is of very frequent occurrence in cardiosclerosis.

In the course of rheumatic fever and during convalescence from this disease any systolic murmur should be regarded as evidence of mitral insufficiency of valvular origin, no matter how faint it may be until its disappearance proves it was of functional or muscular origin.

Tricuspid Insufficiency

The tricuspid valve is rarely the seat of inflammatory lesions. In most cases of endocarditis the aortic and mitral valves are diseased and tricuspid valve is left intact. There are two forms of tricuspid insufficiency:—First, that due to organic valve disease, and second, the muscular or functional insufficiency which is ascribed to excessive dilatation of the right heart. It is supposed to occur in all forms of heart disease after the efficiency of the right ventricle has been impaired, but is especially common in severe mitral stenosis.

Physiology.—Very little is definitely known regarding tricuspid insufficiency. For many years it was held that regurgitation easily occurred at this orifice and that it acted as a factor of safety by lowering the pressure in the right ventricle and protecting it from excessive dilatation.

This theory was advanced by King in 1837 who spoke of tricuspid regurgitation as the safety valve function of the right ventricle. Experimental studies do not support this view for in animals the right ventricle may be distended under great pressure, amounting to 200 or 250 mm. of water during diastole, without causing any regurgitation. (Wiggers.)

In experimental tricuspid insufficiency the mean pressure in the pulmonary artery may fall slightly but the systolic pressure is not affected unless the lesion is severe. (Rihl, MacCallum.) Experimental tricuspid insufficiency leads to hypertrophy and dilatation of the right ventricle (Stadler).

The blood ejected from the right ventricle dilates the right auricle and causes a back flow into the great veins. The weak auricle can compensate but little in overcoming this disturbance of the circulation even when it undergoes hypertrophy. The jugular veins may become distended and pulsate forcibly. Great congestion of the liver often occurs from the regurgitation, leading to necrosis of the central portion of the acini. In chronic cases the primary lesion is replaced by connective tissue and cardiac cirrhosis of the liver results. The amount of blood that flows back into the liver may probably be considerable and the liver has been found to increase its diameter about 1 cm. with each systole (Gerhardt).

The pressure may be very high in the veins near the heart. In one of Gerhardt's cases a 300 gram weight was required to obliterate the pulsation in the jugular vein while the venous pressure in the peripheral veins measured by the method of Moritz and v. Tabora was normal The venous pressure in the arm may reach 25 cm. of water when cardiac weakness is extreme (Hooker).

Physical Signs.—The objective symptoms are not definite nor is their interpretation clear. This statement is easily supported by reference to the venous pulse. It is usually thought that a positive or ventricular form of venous pulse is evidence of tricuspid regurgitation. Instead of the drop in the curve after the *c* wave as normally occurs the elevation is maintained or in place of the *c* wave is a large wave filling the systolic period which is regarded as a *v* wave beginning early in systole. The ventricular form of venous pulse according to Mackenzie occurs only in auricular fibrillation. Wenckebach writing in 1914, said he had seen only three cases of this type of pulse when the rhythm was regular. It seems improbable that tricuspid regurgitation occurs only in auricular fibrillation. In auricular fibrillation the venous pulse is always of the ventricular or positive form and yet in many of them the cardiac efficiency is good and there is no evidence of dilatation of the right auricle as shown by the orthodiagram or on percussion.

Hence it follows that a ventricular type of venous pulse is not diagnostic of tricuspid insufficiency. Mackenzie published years ago the venous pulse tracing from a case of marked tricuspid insufficiency of organic origin, as proved by autopsy, in which the *v* wave was small and the *a* wave by far the highest of the three.

Hering, in 1912 on the basis of experimental work, denied that tricuspid insufficiency occurred clinically. Although this view cannot be entertained it does indicate a lack of accurate knowledge of the subject.

Optical tracings from the jugular in a case of supposed tricuspid insufficiency shows a rapid rise of the curve early in systole. (Fig. 121.)

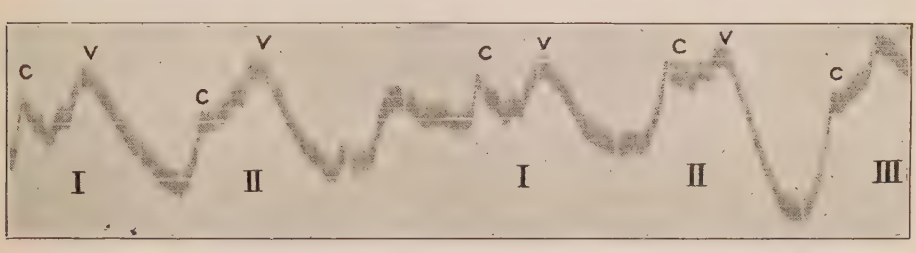


Fig. 121.—Supraclavicular venous tracing by the optical method showing various forms of systolic waves during tricuspid insufficiency. I, prominent v wave beginning to rise early in systole; II, sustained or rising top with murmur vibrations throughout systole. Case of auricular fibrillation. (Wiggers, "Modern Aspects of the Circulation in Health and Disease," Lea and Febiger.)

In tricuspid insufficiency the heart is enlarged to the right and it seems probable that the greater the dilatation of the right auricle the more probable is the existence of a tricuspid regurgitation. I studied a case in which the right border of the heart in a telefluorogram and teleradiogram was in the right parasternal line and 7 cm. from the median line. Yet the jugular veins were not distended nor did the liver pulsate.

A systolic murmur is usually heard over the tricuspid area but it has no distinctive character in relative insufficiency and murmurs transmitted from other orifices are often well heard over the lower end of the sternum. In some cases when the heart is weak or the tricuspid insufficiency of very high degree no murmur may be present. In the much rarer forms of insufficiency due to chronic endocarditis of the tricuspid valve, the systolic murmur is said to be superficial, high pitched, long and rough with the point of maximum loudness to the left rather than to the right of the sternum, and accompanied by a thrill (White). With the development of this valvular lesion the second pulmonic sound frequently becomes weaker.

Diagnosis.—The presence of an expansile liver pulsation, and of a forcible jugular pulse both of the positive or systolic type justifies the

diagnosis of tricuspid insufficiency. It is secondary to severe mitral disease and is associated with other evidence of weakness of the heart muscle. The cardiac dullness is increased to the right of the sternum. There is usually a systolic murmur heard with maximum intensity at the tricuspid orifice. In a case of mitral stenosis, a large hard liver that is not tender, is suggestive of tricuspid insufficiency even in the absence of hepatic pulsation.

Tricuspid Stenosis

This is a rare disease, due to rheumatic endocarditis. At Guy's Hospital, 112 cases were found in a series of 18,000 autopsies, extending over a period of thirty-five years (Pitt). It occurs about four times as frequently in women as in men, and is usually combined with aortic and mitral disease. In fourteen of 195 cases collected from the literature by Futcher the tricuspid valve alone was involved. The stenosis is always associated with insufficiency according to Romberg. Persistent cyanosis, extreme dyspnea, ascites and subcutaneous edema, a presystolic murmur over the tricuspid area differing in character from the mitral presystolic murmur are characteristic signs. Dyspnea may be absent as in a case diagnosed during life by W. W. Herrick. Mackenzie states that the presystolic murmur is rarely heard. It was absent in seven out of Gerhardt's eight cases. Most cases are likely to have auricular fibrillation and this may explain the absence of the murmur in them, but in a case of tricuspid stenosis with regular rhythm in which I made the diagnosis at the autopsy table no murmur was present over the heart. The hypertrophied right auricle may send back a large wave into the jugular with such force as to cause a clear sharp sound preceding the first sound of the heart (Mackensie). The *P* wave in the electrocardiogram should be increased in size. There may be a well-marked liver pulse of the auricular type which although suggesting the presence of tricuspid stenosis may occur in the absence of this disease (Turnbull and Weil). If auricular fibrillation had developed, the liver pulse would be of the ventricular type. One of the striking features of three cases of tricuspid stenosis observed by Levine was the enlarged non-pulsating liver in the absence of cardiac failure and of alcoholic cirrhosis.

Pulmonary Insufficiency

Endocarditis of the pulmonary valve is very uncommon. It is almost always associated with disease of the mitral and aortic valve, and due

to rheumatic infection. Acute inflammation of the pulmonary valve alone has been caused most frequently by the gonococcus. In nine out of a series of twenty-nine cases of pulmonary insufficiency collected from the literature the aortic valve was also involved. The recognition of the pulmonary insufficiency during life when combined with aortic insufficiency is very improbable.

Relative insufficiency with intact valves has been observed in kyphoscoliosis, in the rare cases of narrowing of the pulmonary artery or its branches, and in mitral stenosis. The occurrence of relative pulmonary insufficiency in mitral stenosis has already been discussed; see comment on the Graham Steell murmur, page 472. The physical signs are those of hypertrophy and dilatation of the right ventricle. There may be dullness along the upper part of the left sternal border and prominence of the pulmonary artery in the orthodiagram. The diastolic murmur at the pulmonary area can rarely be distinguished from that of aortic insufficiency.

Pulmonary Stenosis

Stenosis of the pulmonary orifice is rarely observed and is almost always of congenital origin. The cusps of the valve are usually fused together leaving a small narrow opening. Only two cases of acquired stenosis were found by Gerhardt in the recent literature. In differential diagnosis a thrill over the pulmonary area is the only trustworthy sign. The second sound is weak or absent. The electrocardiogram shows a right-sided preponderance which distinguishes it from aortic stenosis. Little importance can be attached to a systolic murmur in this location as accidental systolic murmurs are very common. Balfour called it the region of auscultatory romance. Compression of the pulmonary artery by pericardial or mediastinal bands can give rise to loud murmurs which may be accompanied by a thrill.

Combined Valvular Lesions

It would be a mistake to suppose that patients with valvular heart disease always or even usually present the simple and clear-cut pictures described above under the single lesions. The diagnosis is complicated in about fifty per cent of the cases seen clinically by the existence of more than one valvular lesion. There may be both stenosis and insufficiency of one valve or a lesion of two or more valves. At the mitral orifice

stenosis and insufficiency are usually associated. It is evident that a mitral valve when so thickened and deformed as to produce a stenosis would usually be unable to close so perfectly as to prevent any regurgitation. It is a well-known fact that pure mitral stenosis is rare, although the more marked the stenosis the less the regurgitation. The inflammatory process in a rheumatic infection frequently spreads from the anterior mitral segment to the aortic cusp that lies close beside it. The tricuspid is much more rarely involved in mitral endocarditis and the pulmonary valve is almost immune in rheumatic heart diseases. The combination of aortic insufficiency and aortic stenosis is rare. But when aortic stenosis does develop, there is usually a certain amount of insufficiency associated with it. Relative insufficiency of the tricuspid valves due to congestive dilatation and weakness of the right ventricle is a common sequence of mitral disease. Aortic insufficiency leads to the production of relative mitral insufficiency, when the dilatation of the left ventricle reaches a certain grade. In pure mitral stenosis there is no hypertrophy of the left ventricle but when combined with mitral insufficiency or aortic insufficiency, hypertrophy and dilatation of the left ventricle occur. If tricuspid stenosis is associated with mitral stenosis there may be little or no hypertrophy of either ventricle.

In combined valvular disease, the physical signs of the different lesions may occasionally be distinct and easily recognized as the sum of the signs in the single lesions. More often the signs are modified or obscured, and one valvular defect may lessen the effect of another lesion. When stenosis and insufficiency affect the same valve they may tend to neutralize each other. The more the orifice is narrowed the less blood can regurgitate through it. When aortic stenosis is added to aortic insufficiency the pulse loses its collapsing quality and the pulse pressure becomes small. The diagnostic rule to be followed is to seek first the chief valvular defect. When this is recognized and the signs dependent upon it clearly distinguished, then and only then should the examiner proceed to locate the other valvular lesions that may be present.

Auscultation is the physical method that offers the greatest aid in the diagnosis of combined lesions. A diastolic murmur over the base of the heart is almost always due to aortic insufficiency. This diagnosis can be made with much certainty no matter what other lesions may be combined with it. A systolic murmur due to mitral insufficiency is easily distinguished from that caused by aortic stenosis and when the two conditions exist the combination offers little difficulty in diagnosis. In regard to the frequency of relative tricuspid regurgitation, authorities differ but all agree that it rarely exists except in association with mitral

disease. In simple mitral regurgitation the pulmonic second sound is markedly accentuated. Weakening of this previously loud sound is often a diagnostic sign of developing tricuspid insufficiency (Leube). It is important to note over what valvular areas the different murmurs are heard with maximum intensity. Differences in the character and transmission of the murmurs are helpful in diagnosis. The occurrence of an accentuated pulmonary second sound in aortic insufficiency favors the diagnosis of a mitral lesion combined with it.

Palpation is a distinct aid in some instances. In a case of aortic insufficiency a visible and palpable impulse to the left of the sternum between the third and fifth costal cartilages accompanied sometimes with a diastolic shock over the pulmonary area is to be regarded as an indication of hypertrophy and dilatation of the right ventricle due to associated disease of the mitral valve.

The diagnosis of combined lesions is often difficult and unsatisfactory. "As a rule, the physician is in a safer position if he limits his diagnostic ambition to two valves. Clinically when lesions of three or four valves are determined with accuracy, mortifying post-mortem disclosures are not unlikely to follow." (Osler and Gibson.)

The physician takes the wisest course in most cases when he diagnoses with certainty the chief lesion and leaves the remainder of his diagnosis, as Leube advises, *in suspenso.*

Congenital Heart Disease

Anomalies of development and endocarditis in fetal life are the two causes of congenital heart disease. Commonly these processes are combined, as when a malformed valve becomes the seat of an inflammatory lesion. The right heart is almost invariably involved.

Small defects of the auricular and even of the ventricular septum can exist without producing symptoms or signs. The foramen ovale is often found open at autopsy in cases which have had no disturbance of the circulation.

The most common lesions are pulmonary stenosis and a defect in the upper portion of the ventricular septum. Not infrequently these two pathological conditions are combined.

Pulmonary Stenosis.—The obstruction may be situated in the region of the conus arteriosus of the right ventricle, or at the valvular orifice due to fusion of the segments, or beyond the valve involving the pulmonary artery. The development of children in whom this lesion exists

is markedly retarded. They are often very cyanotic. The skin may assume a dark blue color (*Morbus coeruleus*). The fingers are clubbed and the nails curved. The axillary and suprapubic hairs are scanty. The signs of pulmonary stenosis have been described in the preceding section.

Pulmonary stenosis is frequently combined with other anomalies among which the most frequent are (1) defect of the septum, (2) patent ductus arteriosus and (3) transposition of the great vessels, a condition in which the pulmonary artery arises from the left and the aorta from the right ventricle.

Ventricular Septum Defect.—In 1879 H. Roger described the clinical picture of uncomplicated septum defect which is called by the French the *maladie de Roger*. A characteristic sign of this is a loud harsh long drawn-out murmur which begins with systole and obliterates both heart-sounds. The murmur is not transmitted into the great vessels and is heard with maximum intensity in the middle of the cardiac area, that is, at the left border of the sternum below the third rib. There should be no diastolic murmur as in diastole the curtains of the mitral and tricuspid valves close the opening in the ventricular wall (Reiss). The murmur fills the entire systolic period and unlike the mitral murmurs maintains a constant intensity. A typical thrill is present in most of the cases. Its localization is limited to the third and fourth left interspaces near the sternum. There may be no hypertrophy or dilatation of the heart. In fact the absence of all physical signs of heart disease except the thrill and murmur is of importance in the recognition of this condition. Usually, however, there is moderate enlargement of the heart to the right, more rarely to the left also. Cyanosis is usually absent. The smaller septum defects, which are the most common, often produce no symptoms and hence are generally discovered when a patient seeks relief for some other condition. Careful search has shown that this condition is not very uncommon. One investigator, Hermann Müller of Zurich, has personally studied 47 cases. For a good discussion of this interesting subject the reader is referred to Blumenfeldt's recent monograph in the Kraus-Brugsch System.

Patent Ductus Arteriosus.—The pressure in the pulmonary artery is increased as blood enters this vessel from the aorta, and the pulmonary artery is dilated. This causes a ribbon-shaped zone of dullness to the left of the sternum from the third rib upwards and a bulging of the second arc of the left border of the heart on roentgenological examination. The second pulmonic sound is accentuated, and the right ventricle hypertrophied. A loud harsh murmur which may be continuous is present in the second and third left interspaces. A thrill, usually systolic but some-

times continuous, was present in seven out of nineteen cases collected by Maude Abbott. Its transmission obliquely upward from the second left interspace is regarded as pathognomonic.

Diagnosis of Congenital Heart Disease.—Persistent cyanosis in a

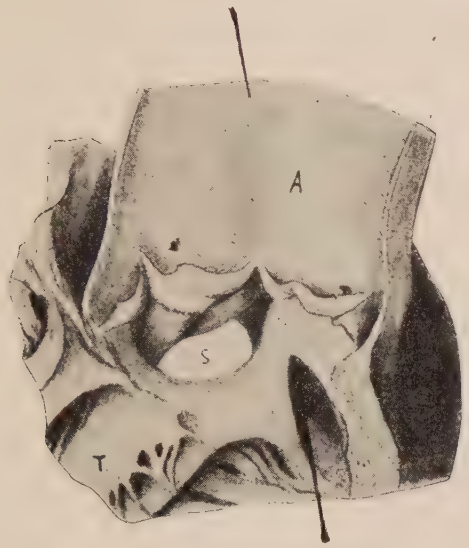


FIG. 122.—Defect (S) in septum ventriculorum, seen from the right ventricle. (A) Aorta. (P) The stenosed pulmonary artery is indicated by a probe which passes through it. (T) Tricuspid valve. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," F. C. W. Vogel.)

child together with the finding of a loud rough murmur over the heart is sufficient for the diagnosis of congenital heart disease. The lesions are often multiple and complicated and their precise nature cannot usually be determined. There may be no murmur or no cardiac enlargement and yet some anomaly be present. The examining physician is frequently forced to the conclusion that while the patient has some form of congenital heart disease an exact diagnosis is impossible.

Cardiosclerosis. The Senile Heart

This is the most common form of heart disease. In a series of 230 cases seen in my private practice, 139 were chronic myocardial disease. On analyzing all cases of chronic organic heart disease without evidence of valvular lesions in this series the relation of primary myocardial disease to age is at once apparent.

TABLE V

CHRONIC MYOCARDIAL DISEASE

Age	Number of cases
30–40 years	1
40–50 "	17
50–60 "	45
60–70 "	51
70–80 "	22
80–90 "	3
	139

The nature of this chronic myocardial disease is not clear in many details, but its relation to age and to arteriosclerosis in general, is undoubted. The senile heart is the most accurate designation to apply to this clinical group in the present imperfect state of knowledge of its pathogenesis. The term cardiosclerosis or myocardial degeneration is preferable to fibrous myocarditis, because freqeuntly no fibrosis can be demonstrated at the autopsy table or on histological examination of the heart muscle, and also, because typical fibrous myocarditis may be the end result of rheumatic heart disease and hence belong in a different group etiologically. There is a similar objection to grouping these cases under the heading coronary endarteritis as is done by Krehl and Romberg. Although it is highly probable that anatomical or functional disease of these arteries is the cause of the senile heart there is no evidence of this in many cases as the coronary arteries even in their smaller branches appear healthy.

The designation chronic myocarditis is objectionable because, first, the underlying pathological process is a degeneration and not an inflammation and secondly, it leads to confusion as the name chronic myocarditis is correctly limited by Krehl and others to the chronic changes in the heart muscle that result from the action of the bacteria or toxins of infectious diseases, particularly rheumatic fever, see page 487. In some classifications, the heart in cases of hypertension, whether of the essential type or that occurring in chronic Bright's disease, is not included under arteriosclerotic heart disease, but placed in a separate group. The clinical features exhibited by the hearts in hypertonia are similar to those with normal blood-pressure. In many cases sclerosis of the renal arteries or of the coronaries themselves is probably the chief causative factor in the cardio-

pathy of hypertensive disease. I have, in the past, placed the cases with high blood-pressure in this cardiosclerotic group, but I agree with White that it is better to consider them separately at least for the present. The term hypertensive heart disease is descriptive, and as the cause of the heart changes in hypertonia is unknown, it has the advantage over "cardiosclerosis with hypertonia" of not committing one to any theory regarding the etiology.

The symptoms of the senile heart are those of cardiac insufficiency in general (see page 449), and in addition two symptom complexes are common. These are *angina pectoris* and *cardiac asthma*. The pain in angina is usually substernal and brought on by exertion. It may vary from a mild oppression to agonizing pain. Cases of substernal pressure are much more common than severe pain. The pain frequently radiates to the left arm, sometimes to the right arm, neck or back. There were fifty-nine cases classed as angina in my series of 139 cases. Cardiac asthma occurred in fifteen of my cases. The condition consists of nocturnal attacks of dyspnea usually coming on after the patient has been quietly sleeping for several hours. The usual duration of an attack is fifteen minutes to an hour. It is sometimes the first symptom of cardiac insufficiency but is always a sign of grave impairment of the myocardium.

Cyanosis is unusual even in severe heart failure due to cardiosclerosis. There is often slight chronic passive congestion of the lungs as shown by dullness and moist râles located most frequently over the bases posteriorly. The heart is often markedly enlarged. Often, however, the cardiac impulse cannot be seen or felt even when there is great hypertrophy and dilatation. There may be enlargement of the left ventricle only, but in most cases both ventricles are found hypertrophied and dilated at autopsy. The cardiac outline on percussion, in a goodly percentage of the cases, is increased to the right as well as to the left. The heart-sounds may be clear, but are often enfeebled. The second aortic sound is usually somewhat accentuated, due more often to sclerosis of the aorta than to high blood-pressure. There is frequently a systolic murmur at the apex, the result of dilatation of the muscular ring that surrounds the mitral orifice. This is usually soft and blowing, but may be harsh and loud. It may appear long before there are any signs of cardiac weakness. It is often not constant, but is heard at one examination and absent at the next. It follows no rule. It may be present when the heart action is strong and disappear with the onset of heart failure, or it may only be heard during a period of marked cardiac insufficiency. On the basis of this murmur these cases in the past have often been mistaken for mitral insufficiency of

valvular origin. A systolic murmur at the aortic area is common. Auricular fibrillation is of frequent occurrence, as are also the various forms of heart-block (see page 430).

Teleradiograms and especially telefluorograms are of great value in the diagnosis of cardiosclerosis. (See page 402). The movements of the ventricles seen on the fluoroscopic screen even when the heart is greatly hypertrophied are usually slight. The contrast between the slight motion of the heart in cardiosclerosis and the vigorous contraction of the heart in aortic insufficiency of rheumatic origin and of the thyroid heart is very striking. Dilatation of the aorta is common and the shadow seen in the first oblique diameter is thick and the upper end clubbed.

Coronary Thrombosis. Infarction of the Heart

The symptoms resemble those of angina pectoris, but the agonizing pain is of longer duration, lasting for hours and days. The onset is sudden. The face assumes a pasty ashen pallor. The location of the pain is often substernal, but may be in the epigastrium. The cardiac impulse cannot be felt, the heart is enlarged on percussion, and the sounds are weak. The pulse is rapid and feeble. Dyspnea is a common symptom and is severe. It was present in sixteen out of nineteen cases studied by Wearn. Fine moist râles are almost constantly present over the lungs. When the pain is in the epigastrium the condition may be mistaken for an acute abdominal inflammation (Levine and Tranter). There may be muscular rigidity and tenderness over the upper abdomen. A leucocytosis is of common occurrence. (Wearn.) In one case there were 33,500 leucocytes per cmm. (Levine and Tranter.) Fever is common and usually reaches 101° or 102°. The liver is frequently enlarged and tender. There may be a transitory pericardial friction-rub. Gorham has emphasized the importance of this diagnostic sign. The clinical features of the disease have been well described by Herrick in a series of papers.

In distinguishing coronary thrombosis from ordinary angina pectoris, Clifford Allbutt emphasizes "the flutter and lability of the heart, the rapid, irregular and failing pulse, the waning sounds, the dyspnea and the cyanosis." Furthermore, the blood pressure falls, while in angina it rises or remains stationary.

Rupture of the Heart

A healthy heart muscle probably never ruptures as a result of strain. The common cause of a break in the ventricular wall is cardiac infarction,

due to closure of the coronary artery by a thrombus or embolus. A cardiac aneurism may first form at the site of the infarcation. This is a localized bulging of the weakened heart wall occurring as a result of the high intraventricular pressure. Later this aneurism breaks at its weakest spot. The condition occurs most frequently in men between sixty and sixty-five affected with sclerosis of the coronary arteries. It is only when sclerosis of the coronaries exists that thrombosis of a branch of the main artery leads to infarction.

The occurrence of rupture in a portion of the wall that has undergone fatty change has been repeatedly observed since Quain first called attention to it. Fatty degeneration is not the cause of the rupture. It is simply the result of a lack of nourishment supplied to the affected muscle fibres by the occlusion of the branches of the coronary arteries that feed them with blood.

The escape of blood from the ventricle into the pericardium, resulting from rupture of the heart, produces the condition known as *hemopericardium*. The more rapid the escape of blood, the quicker will death occur and the less the amount of blood found in the pericardium at autopsy. If there is only a minute opening in the heart wall, effused blood will collect slowly and the sac become greatly distended. Such cases may live several days and the physical signs will then be those of pericardial effusion with the general symptoms of internal hemorrhage.

A series of attacks of angina pectoris may precede the fatal issue by hours or even days. If the course of the disease is protracted a pericardial rub may sometimes be heard over the infarcted area. The cases of rupture in which life is sufficiently prolonged to permit of the development of physical signs are rare. Usually death quickly follows the rupture and the diagnosis is not possible during life.

Chronic Myocarditis

Enlargement of the heart due to hypertrophy and dilatation can occur as the end result of rheumatic infection apart from any valvular lesion. In time symptoms of cardiac weakness usually develop. The condition is rare and its recognition even more rare. The diagnosis of chronic myocarditis is rendered probable by the discovery of a permanent disturbance of conduction (heart-block) in a patient below the age of thirty-five who gives a history of rheumatic fever. Most of the cases are mistaken for mitral insufficiency of valvular origin. A systolic murmur at the apex and the existence of definite cardiac enlargement naturally has led to this diagnosis. The possibility that even loud mitral murmurs may be due to

muscular insufficiency of the mitral orifice is not yet generally known. If a murmur is present in a case of cardiac enlargement the existence of mitral endocarditis cannot be positively excluded during life. Deaths in chronic myocarditis without valvular disease must be uncommon or they would be more frequently seen at autopsy. The reason for this apparent rarity of chronic rheumatic myocarditis is the involvement of the valves in the vast majority of the cases of rheumatic heart disease. There can be no doubt that chronic rheumatic myocarditis is a very common and serious condition, but it is usually associated with mitral stenosis or aortic insufficiency. A slight mitral stenosis, the result of a mild endocarditis, rivets the attention of both clinician and pathologist and the hypertrophy and dilatation of the ventricles are regarded as secondary to the valvular lesion. The truth is that the primary disease of the heart muscle has been the real cause of cardiac failure. In the cases of moderate mitral stenosis when symptoms of heart failure develop they usually result from the damage inflicted on the myocardium by the rheumatic infection. In these cases, death is really due not to the valvular lesion, but to chronic myocarditis.

Few proven cases of chronic rheumatic myocarditis without valvular lesions have been reported. Berry has recently made a complete study of one such case that was diagnosed during life. The patient was twenty-eight years old. There were symptoms of severe cardiac weakness, and the electrocardiogram showed a right bundle branch block. He had had rheumatic fever eight years before the cardiac symptoms developed. He died during an attack of bronchopneumonia. The heart weighed 740 grams. On gross and microscopic examination the heart muscle presented the ordinary picture of fibrous myocarditis.

Only in young people can the condition be distinguished from cardiosclerosis, as the symptoms and physical signs may be the same in both conditions. In acute rheumatic myocarditis, the perivascular collections of cells known as Aschoff's bodies are constantly present, but in chronic myocarditis they are constantly absent (Coombs).

In the recognition of chronic rheumatic myocarditis the discovery of delayed conduction through the bundle of His or partial heart-block in a young person is invaluable. Polygrams or preferably electrocardiograms should be obtained if possible in all suspected cases.

Endocarditis Lenta or Subacute Infectious Endocarditis

This is the one form of endocarditis with a definite clinical picture. Unlike rheumatic infection which attacks the myocardium as well the en-

docardium, the inflammation in endocarditis lenta is limited to the heart valves, and the lining of the cavities. It produces a long continued septicemia that is almost invariably fatal. The duration is frequently a year or more. The diagnosis is rarely made in an early stage even by experienced physicians owing to the lack of localizing symptoms. The disease affects men more frequently than women, and the majority of cases occur between the twentieth and fiftieth year. The disease may be due to different bacteria, occasionally the influenza bacillus and streptococcus hemolyticus but usually streptococcus viridans. On blood agar plates this microörganism produces small greenish colonies which do not hemolyze the blood. In more than half the cases, the disease attacks those who have already damaged heart valves, the result usually of a previous rheumatic infection, but occasionally it develops on the site of syphilitic aortic valve lesions. The left side of the heart is usually involved, the mitral more often than the aortic valve. The thrombi on the valves form large cauliflower-like growths which often involve the cordae, tendinae and the mural endocardium. At the onset it has sometimes been mistaken for influenza and later for pulmonary tuberculosis. The fever is mild and of a remitting type, and there are pains in the back and limbs. The temperature at times may rise to a maximum of 101° or 102°. The only other general symptoms are loss of weight and increasing anemia. The nature of the disease and the involvement of the heart valves is often not recognized until a blood culture is made and streptococcus viridans isolated. Positive blood cultures are obtained in over ninety per cent of the cases (Libman).

For a long period the examination of the heart may reveal no evidence of disease in those patients who have not had a previous rheumatic endocarditis. The size of the heart is not increased in the early stages, and although a systolic murmur is frequent, there may be no murmur present even in advanced endocarditis. When the disease develops in an old rheumatic heart a new murmur often appears. As the disease advances cardiac enlargement can usually be detected by percussion. For a long time after the diagnosis has been made, the cardiac reserve may apparently be but little if any impaired. One of my patients after the disease had existed some time had a vital capacity of 5000 cc., which is a high normal.

There is a continuous tachycardia which is out of proportion to the low grade of fever. Embolic manifestations form the most characteristic feature of the disease. Infarcts often form in the spleen and kidney. The urine frequently contains red blood corpuscles on microscopic examination of the sediment and this finding points to the existence of an embolic focal nephritis. Small hemorrhages in the skin from the size

of a pinhead to a split pea are common. The petechiae often have white centers. The sternum may be tender (Libman). Painful subcutaneous nodules on the terminal phalanges of the fingers and toes often develop. (Osler.) The fingers often become clubbed as the disease progresses. (Fig. 123.) In fact clubbing of the fingers is one of the most valuable

Fig. 123.—Clubbed fingers in subacute bacterial endocarditis. Note the discoloration of distal phalanges due to hyperemia. (Dr. Randolph R. Byers.)

diagnostic signs in this disease (Cotton). It is attributed by Leschke to a toxic alteration of the distal tissues and by Cotton to local venous stasis. There is a peculiar pigmentation of the face (Libman). Death results from embolism, nephritis, sepsis, or cardiac insufficiency.

Septic Endocarditis. Acute Bacterial Endocarditis

The malignant endocarditis of the older writers runs a much more rapidly fatal course than the more common subacute endocarditis. Septic endocarditis is caused by a number of microörganisms including the influenza bacillus, streptococcus hemolyticus, staphylococcus aureus, and gonococcus. The clinical picture is not that of heart disease, but of septicemia.

The Thyreotoxic Heart

In cases of well developed exophthalmic goitre the heart is usually hypertrophied. If the thyroid disease progresses the heart becomes insufficient. In milder cases of hyperthyroidism the cardiac symptoms may dominate the clinical picture. The cardiac insufficiency may be marked when only slight cardiac enlargement exists, and must be attributed to a functional injury of the heart by the internal secretion of the thyroid. Anatomical alterations were not found in some carefully studied cases (Hezel). Others showed sclerotic lesions in the heart valves and coronary arteries. Severe cardiac weakness sometimes develops in susceptible

persons after taking iodine even in small amounts. This has long been recognized in some regions such as Switzerland where endemic goitre exists.

The disturbance of the heart may consist simply of an unpleasant excitability consisting of periods of forcible and rapid heart action, or there may be a continuous tachycardia with pounding of the heart. Systolic murmurs are often present over the mitral, pulmonary and aortic areas. The aortic second sound is accentuated and the first sound at the mitral area may be unusually loud and sharp. Both the paroxysmal and continuous types of auricular fibrillation are common. The aorta is sometimes found dilated when the heart is examined by the orthodiagraph in the first oblique diameter.

For the diagnosis the demonstration of some of the other thyreotoxic disturbances is essential. These are an enlarged thyroid gland, exophthalmos, rapid tremor, loss of weight, diarrhea, and high basal metabolism. The orthodiagram shows a heart of typical mitral form. The cases are frequently mistaken for mitral insufficiency of valvular origin. The systolic murmur at the aortic area together with the accentuated second sound and the dilatation of the aorta demonstrated by the x-ray may be wrongly interpreted as signs of aortic sclerosis. In the recognition of the thyreotoxic heart the determination of the basal metabolism has proved of great value.

CHAPTER VIII

DIAGNOSIS OF THE CARDIAC NEUROSES

Cases of neurasthenia with symptoms referable to the heart are very common and are often mistaken for organic heart disease. As the disease is in the mind of the patient and not in his heart these cases belong in the domain of neuropsychiatry. White holds that the introspection neurosis (hypochondria) is the most common type with cardiac symptoms, and that the fatigue neurosis includes most of those not included in the first group. As in all neurotic conditions the subjective symptoms are rich and varied while the physical signs are slight. The patients complain of pounding of the heart (palpitation), pain in the region of the heart—the usual situation being below the left nipple, rarely substernal,—and dyspnea on exertion or even at rest. Speedy fatigue on exertion is frequent. Vasomotor disturbances are common. These give rise to dizziness, faintness, flushing, pallor, sweating, sense of heat or chilliness, coldness of hands and feet.

The patients often have a tense anxious look. There is frequently pallor of the face, but sometimes especially in women, the face and neck and a part of the chest may be deeply flushed (erythema). The lips may be cyanosed in severe cases. More often the hands are blue and clammy. Sweat may drip from the axillae.

The pulse-rate is usually rapid. It is rarely less than 100 during the examination. After slight exertion or during excitement it increases unduly. Occasionally the pulse may be slow (vagotonia), and the bradycardia may be increased by emotional disturbance.

Respiratory arrythmia is common in young persons. Extrasystoles are not infrequent and often cause of disagreeable sensations. The blood-pressure is labile and often temporarily increased. Rarely the blood-pressure may be elevated for weeks or months, at least at the time of the examination. The cardiac impulse may be visible over a wide area. It is increased in force but is not heaving. A pseudothrill is sometimes felt at the apex. Hyperesthesia over the apex region is common in women patients. The percussion outline is not increased. The heart-sounds are loud and the first sound sharp at the apex. There may be a reduplication and impurity of the first sound that is easily mistaken for a presystolic

murmur. The heart-sounds are usually clear but a systolic murmur may be present especially at the apex. The vital capacity is usually normal but may be slightly lowered.

Caution should be exercised in basing the diagnosis of cardiac neurosis on normal heart findings in patients above the age of forty who complain of pain in the cardiac region. Often angina pectoris due to disease of the aorta or coronary arteries occurs when nothing abnormal can be detected on the physical examination. Hyperthyroidism in the absence of a distinct goitre and of exophthalmos may easily be mistaken for a cardiac neurosis. Cardiac neurosis often takes its origin from a mistake on the part of the physician who bases a diagnosis of heart disease on a systolic murmur, a physiological sinus arrythmia, extrasystoles or other inconsequential findings.

CHAPTER IX

DIAGNOSIS OF DISEASES OF THE PERICARDIUM

Acute Pericarditis

This occurs chiefly in association with three diseases,—rheumatic fever, pneumonia and chronic nephritis. The involvement that results from tuberculosis is considered by itself as it is more chronic and has special characteristics. Rheumatic infection is the most common form of acute pericarditis that is recognized clinically. In pneumonia an inflammation of the pericardium that produces no symptoms and physical signs is often discovered at necropsy.

In the first stage of acute pericarditis there is a deposit of fibrin on the surface of the pericardium (fibrinous pericarditis). It may be small in extent and consist of fine granules, giving the serous surfaces the appearance of sandpaper, or it may be extensive, after the disease has lasted some time, and then the fibrinous masses by the movement of the heart are arranged in shreds and strings forming a shaggy coating (cor villosum). A serous exudate is poured out in varying amounts giving rise to serofibrinous pericarditis. Other pathological forms of acute inflammation are the hemorrhagic and suppurative. In acute rheumatic pericarditis the exudate may measure 600 cc. or more, but the amount is almost never so great as to endanger life and to make parancentesis necessary (Curschmann). In more chronic conditions (tuberculosis), the fluid exudate has measured as much as 4000 cc. (Thayer).

Physical Signs.—The most common and the most trustworthy sign of pericarditis is the friction-rub. This has been described in the section on auscultation. (See page 342.) As the murmur may come and go in the course of a few hours, it is important to listen repeatedly in a case in which pericarditis is suspected. Flint held that it was always present in dry pericarditis, but it is certainly not heard in all cases in spite of frequent careful examinations. Of the thirty-four cases of rheumatic pericarditis studied by Levine and diagnosed during life, it was absent in five, and of twenty-nine cases occurring in nephritis it was not detected in eleven. A large number of cases of acute pericarditis, found at autopsy, especially those occurring in pneumonia and nephritis, have been unrecognized clinically as no friction-sounds were heard. "Constantly keeping in

mind the possibilities of acute pericarditis as a complication in certain diseases (rheumatic fever, pneumonia, Bright's disease and as a terminal event in general septic processes) diminishes the number of cases that are overlooked." (Levine). The friction-rub although usually transitory may be constant and persist for weeks.

As fluid increases in the pericardial sac, the cardiac impulse weakens, see page 282, and the area of cardiac dullness increases, see page 307. The

Fig. 124.—Heart with pericarditis. On the surface of the heart the fibrin appears as fine nodular (z) elevations or as a projecting network. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," F. C. W. Vogel.)

visible impulse is often a considerable distance inside the left border of dullness while in cardiac dilatation, with which pericardial effusion is often confused, the outermost point of visible or palpable impulse is at or near the left border of dullness. In large pericardial effusion, the area of absolute dullness is greatly increased so that its border may coincide with that of the relative dullness. This distinguishes it often from cardiac

enlargement in which a zone one centimeter or more usually separates the borders of relative and absolute dullness.

Changes from day to day in the location and character of the impulse and of the cardiac outline on percussion are of great aid in distinguishing pericardial effusion from cardiac dilatation.

A patch of dullness and bronchial breathing near the angle of the left scapula is of distinct value in the diagnosis of acute pericarditis. (Bamberger's sign, also known as Ewart's sign.) Râles in this area although usually absent may be present and continue to be heard for from one to three weeks. This sign is sometimes found before the friction-rub appears. Christian found it in nearly 15 per cent of his cases. It is stated that this dull area may be reduced in size if the patient bends forward.

Partial heart-block or delayed conduction occurs more often with acute rheumatic pericarditis than in almost any other disease. It is a manifestation of the acute myocarditis that in some degree always accompanies the pericarditis.

The radiogram of the heart and central vessels may be shaped like a gourd bottle or a *"brioche."* Often it is of triangular outline. The normal division of the borders into arcs is obliterated.

Tuberculous Pericarditis

This is of great clinical importance because in a certain proportion of cases the effusion rises to a higher tide, as Clifford Allbutt terms it, than in any other disease. No serious disease is so frequently overlooked as pericarditis with effusion (Osler). The fluid may be hemorrhagic. Tapping of the sac to relieve symptoms is rarely necessary in rheumatic pericarditis, but it is common in tuberculosis pericarditis. Often the fluid recurs and repeated tappings are necessary.

Pneumopericardium

Reference to this very rare condition has already been made, page 310. There is always fluid present, either serum, pus or blood in addition to air. The splashing of the heart causes a continuous gurgling churning sound likened to the wheel of a water mill, and called by the French, *bruit de moulin.* It may be so loud as to disturb a patient's sleep (Sinnhuber). A tympany replaces the normal cardiac dullness, but this and metallic heart-sounds are not sufficient for a diagnosis as they occurred in one of my cases of pneumothorax.

Adhesive Pericarditis

(Adherent pericardium, Chronic mediastinopericarditis, Synechia cordis). When the two layers of the pericardium are adherent, and dense bands unite the sac to the chest wall and diaphragm, the work of the heart is increased and hypertrophy results. The symptoms may be those of cardiac insufficiency. Chronic passive congestion of the liver is common. In other cases pseudohepatic cirrhosis may develop and chronic inflammation of the serous surface of the liver (Zuckergussleber) occur. The combination of adhesive pericarditis and cardiac cirrhosis is known as *Pick's disease*. The cardiac symptoms may be slight and the cases are usually mistaken for alcoholic cirrhosis with ascites. Mention has been made of the systolic retraction of the wall of the chest in this disease, page 283.

There is a palpable diastolic shock in the region of the apex. There may be a murmur heard in diastole. In a series of twenty-four cases of adherent pericardium with no valvular lesions found at autopsy, seven had been diagnosed aortic insufficiency on account of this diastolic murmur associated with cardiac hypertrophy (Foster).

Sibson and more recently Wenckebach noted that in adherent pericardium owing to the fixation of the heart the anterior portion of the chest above the apex impulse moved forward only slightly in inspiration and that the abdomen instead of moving forward was actually retracted. There may be a systolic recession in the left back in the tenth and eleventh intercostal spaces between the scapular line and the vertebral border (Broadbent's sign). A tracheal tug may be present. There may be no shifting of the position of the apex impulse when the patient changes from the right to the left side, but this fixation of the impulse may occasionally be found when no adhesions exist. (Bäumler.) The fluoroscope may show that with the breath held in deep inspiration, the left side of the diaphragm is lifted at each systole (Schwarz). In radiograms the cardiac contour is sometimes marked by projections and depressions.

The pulsus paradoxus may be present in adherent pericardium and occasionally in pericardial effusions. It is of the type that Wenckebach regards as due to mechanical and not to dynamic disturbances of the circulation, see page 441. The pulse waves are small in inspiration, larger in expiration and reach their maximum size during the respiratory pause. When the circulation is markedly embarrassed by mediastinal adhesions the veins in the neck are apt to remain distended even with the patient in the upright position.

The diastolic collapse of the distended jugular veins described by Friedreich is not a trustworthy sign.

CHAPTER X

DIAGNOSIS OF DISEASES OF THE AORTA

Acute Aortitis

The possibility of symptoms due to acute inflammation of the aorta in rheumatic fever, influenza and typhoid fever should be recognized. This condition has been chiefly studied by the French. The best description appears in Allbutt's "Diseases of the Arteries." In America it is rarely recognized, because its existence is not considered. (T. McCrae.) Pain beneath the upper part of the sternum is the chief symptom. It may vary from a sense of tightness to one of agonizing severity. There may be attacks of breathlessness and a sense of oppression in the chest. The physical signs are not characteristic and usually a definite diagnosis cannot be made clinically. There may be an upward displacement of the subclavian arteries above the level of the clavicles (Potain's sign). This is attributed to a lengthening of the ascending aorta. It may also occur in syphilitic aortitis. A pulsation of the aorta may be seen and felt in the aortic notch. There may be dullness over the manubrium especially to the right of the median line. The second sound at the aortic area has a somewhat tympanitic quality that is characteristic. The fluoroscopic examination especially in the first oblique diameter should show dilatation and lengthening of the ascending aorta in well-marked cases of the disease. In distinguishing the acute inflammatory aortitis from the syphilitic form the Wassermann test is necessary.

Syphilitic Aortitis

This is a common late manifestation of syphilis. It is a frequent disease in the large municipal clinics and is very common in the hospitals of the southern states, especially in negroes. It is apparently met with more often in general consultation practice in Germany than in America. It formed 26 per cent of the cases of organic diseases of the heart and aorta that Romberg saw in his private practice, while among 200 consecutive cases of heart or aortic disease, in my practice, less than 2 per cent were syphilitic, although a Wassermann test was a routine procedure.

It usually develops about twenty years after infection. Its occurrence in patients under thirty-five years is rare.

The typical appearance at autopsy is a localized, deeply furrowed or crinkled thickening of the internal surface of the first part of the aorta. Sharp borders separate the diseased area from the smooth normal aorta beyond. The disease is caused by primary inflamation of the media (mesaortitis syphilitica), and the changes in the intima and adventitia are secondary.

The most common subjective symptom is pain behind the manubrium. It may be continuous or only produced by exertion. It often extends from the chest to the left shoulder or left arm. It is due, according to Huchard, to the inflammation of the adventitia, which is rich in nerves. Dyspnea occurs less frequently than pain and is usually the first indication of cardiac insufficiency of the congestive type.

The most important and frequent sign of aortitis is the accentuated ringing aortic second sound. This is usually associated in this disease with a normal blood-pressure. It frequently persists after the development of aortic insufficiency. A systolic murmur at the aortic area is common. It is often soft and faint and is never as loud and rough as the murmur in aortic stenosis. There may be dullness over the manubrium or at the sternal end of the first and second intercostal spaces. Pulsation in the suprasternal notch and in the second right interspace is common in aortitis complicated by aortic insufficiency. A definite diagnosis can rarely be made without the aid of an orthodiagram or teleradiogram, which shows a dilatation and lengthening of the aorta. (See page 402.)

Insufficiency of the aortic valves was present in about 30 per cent of the cases in Romberg's Munich clinic. Angina pectoris developed in 14 per cent and saccular aneurism in 16 per cent. The facies of patients with syphilitic aortic insufficiency has the pallor that has so long been associated with this form of valvular disease, while those with rheumatic aortic disease are often high colored (Blumer). In every case of aortic insufficiency it is imperative that a Wassermann test be performed and in every case of syphilis in which chest pain occurs a careful radiological examination of the aorta should be made.

Aneurism of the Aorta

A circumscribed dilatation of an artery is called an aneurism. The so-called diffuse and fusiform aneurisms are better described as forms of simple dilatation of the aorta. A true aneurism of the aorta, in the sense

in which the word is usually employed in clinical medicine, is called by pathologists a saccular aneurism (aneurysma sacciforme). This is due to a syphilitic aortitis in the vast majority of cases. The statement is usually made in text books that sudden trauma and arteriosclerosis may occasionally cause it. It is more probable, however, that all saccular aneurisms of the aorta that are seen clinically develop from a syphilitic aortitis (Romberg). They are most commonly found in the ascending aorta, and usually form a more or less globular protrusion of the wall, which is connected with the interior of the aorta by a constricted portion called the neck. They are usually the size of an apple, but may attain much larger proportions. The intima is often absent over the wall of the aneurism, but if present it shows the characteristic changes of luetic aortitis. The media is thinned and the adventitia greatly thickened. Thrombi frequently line the inner wall of the cavity and occasionally fill it.

FIG. 125.—Aneurism A, in the first portion of the aorta. The inner surface of the aorta shows the uneven appearance characteristic of syphilitic sclerosis. E is the orifice of the aneurism. (Ribbert, "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," F. C. W. Vogel.)

Those on the free surface are soft, while adherent to the wall are firm older fibrinous layers of yellowish color, that are sometimes calcified.

Pain is the chief symptom. It is chiefly localized behind the sternum, but may radiate to the back, shoulders or arms. Pain of a neuralgic character may result from pressure on the intercostal nerves or brachial

plexus. Dyspnea, cough, often of a brassy quality, and difficulty in swallowing result from pressure of the aneurism on neighboring structures—bronchi, recurrent laryngeal nerve, esophagus. Definite symptoms of cardiac weakness may develop from complicating disease of the heart muscle.

The aneurism is due to the yielding of the weakened aortic wall over the area of localized inflammation (mesaortitis) to the high pressure of the blood in the aorta. Hypertrophy and dilatation of the heart do not occur unless aortic insufficiency or myocardial disease is likewise present.

Physical Signs.—Broadbent made a useful distinction between the aneurisms of symptoms and the aneurisms of physical signs. The former are located in the arch and the descending limb, the latter in the ascending portion just above the aortic valves. In many cases, however, it is impossible to discover in what part of the arch the aneurism takes its origin.

FIG. 126.—Aneurism of the arch of the aorta. Front view. (The collection of Dr. C. N. B. Camac.)

The objective examination should always begin with a careful inspection of both the front and back of the chest, and the patient should be stripped to the waist and placed in a good light. The chest should be inspected from different positions. Slight pulsation may only be visible

when an oblique light falls on the skin. (See page 277.) Many deeply seated aneurisms produce no demonstrable pulsations, in others the abnormal movement is so striking that it is possible to make the diagnosis at a glance. The most frequent location is in the second intercostal space. In aneurisms of the ascending portion of the aorta, it is usually to the right of the sternum and in aneurisms of the arch to the left of the sternum. In the rarer aneurisms of the descending thoracic aorta the pulsation is visible in the left interscapular fossa. When the aneurisms attain a certain size they project from the chest as tumors. The skin over a protruding aneurism is thinned by pressure, and may finally undergo

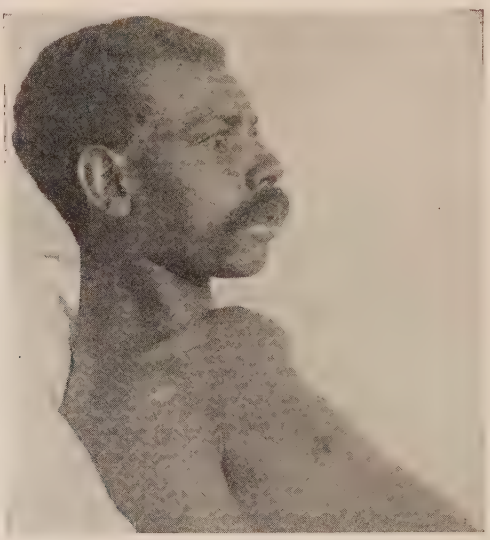


FIG. 127.—Aneurism of the arch of the aorta. Side view of same patient. (The collection of Dr. C. N. B. Camac.)

necrosis. The marked *bulging* of some aneurisms is only made possible by the gradual destruction of the muscles, ribs and sternum by pressure. These aneurismal tumors are recognized by their strong expansile pulsation. Occasionally even large aneurisms pulsate weakly or not at all, owing to the large thrombi in their sacs.

PALPATION.—This yields three important diagnostic signs of aneurisms. (1) A *thrill,* usually systolic in time, is sometimes present and is best felt by the palm of the hand pressed lighty on the tumor. (2) *Diastolic shock.* This is very characteristic of aneurism. It was regarded by Broadbent as absolutely pathognomonic. It is felt on light palpation as a sharp snapping short shock. A systolic shock may occasionally be present. (3) *The expansile pulsation.* Its character is like

that of the cardiac impulse. To learn to recognize it one should familiarize himself with the sensation given to the palpating fingers by an actively beating cardiac apex. (Osler.)

Deep-seated aneurisms may communicate to the chest wall a forcible heave that can best be recognized by placing one hand on the sternum and the other on the middle of the spine and exerting firm pressure. By pressing a finger deep into the suprasternal notch the pulsation of an aneurism arising from the arch can sometimes be felt.

Tracheal tugging. Adhesions of an aneurismal sac to the trachea are apt to produce a slight downward pull of the larynx with each beat of the

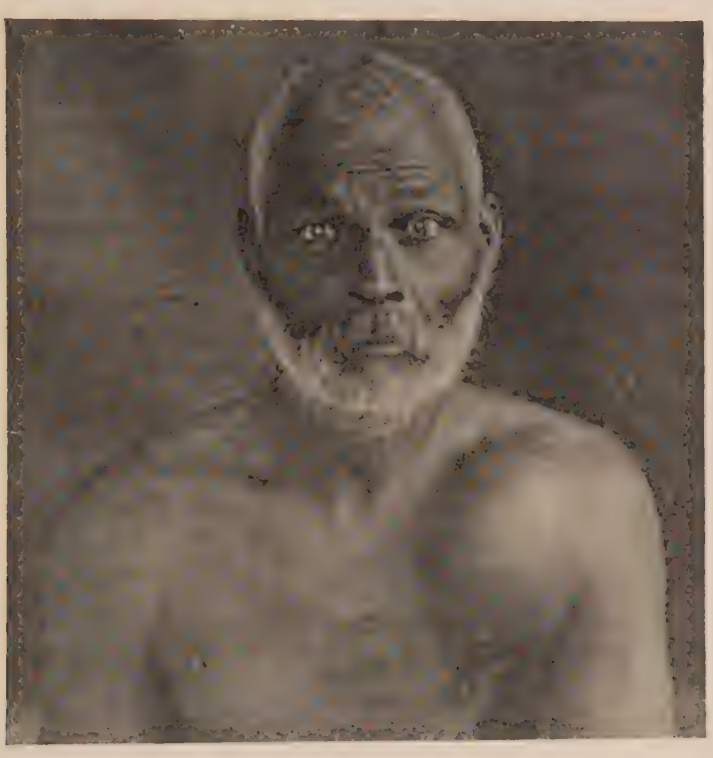


FIG. 128.—Subclavian aneurism. The general position and appearance of the external tumor are similar to that sometimes occurring in aneurism of the arch of the aorta, except that it is further to the left of the median line. (The collection of Dr. C. N. B. Camac.)

heart. The tug is also caused by the transmission of pulsation to the left bronchus and thence to the trachea from aneurisms on the concave side of the arch of the aorta. It is of common occurrence and is of considerable value in diagnosis although it may occur in great dilatation of the aorta, in mediastinal tumors and in chronic mediastinopericarditis.

The patient should be in the erect postion, and the cricoid cartilage grasped by the thumb and forefinger and pressed slightly upward. A tug will be distinctly felt with each pulsation of the aorta if the sign is present.

Inequality of the radial pulse in the two arms and less commonly of the carotid pulse may be present. (See page 349.)

Percussion.—*Dullness* over the aneurism is usually present if the sac is near the sternum, corresponding to the position in which pulsation is most frequent. Dullness may also be present over the manubrium and in the first and second left interspaces, and in the rare cases of aneurism of the descending aorta in the left interscapular fossa. A narrow zone of tympany may extend beyond the dull area or be present without any dullness. (Strümpell.) It is due to retraction of lung-tissue near the aneurismal sac.

Auscultation.—Both heart-sounds are usually well heard. A clear ringing accentuated second sound is the most distinctive auscultatory sound in aneurism. The more remote its maximum intensity from the aortic valves, the greater the significance of this ringing low-pitched second sound. A systolic murmur is common. If aortic insufficiency exists the diastolic murmur is often well heard over the sac. The second sound is rarely replaced by this murmur. If the aneurism is filled with fibrinous masses, no sounds may be heard over it.

The Roentgen examination is of great aid in the diagnosis of aneurism. It frequently leads to the detection of aneurisms that are producing no physical signs. (See page 405.)

Signs of Compression.—Pressure on the veins by an aneurism may lead to swelling of the veins of the neck, arms or skin of the thorax. Direct pressure on the superior vena cava leads to marked cyanosis of the face and neck, prominence of the jugular veins, and edema of the face and arms. Compression of the trachea may cause severe dyspnea, while pressure on the left bronchus causes the symptom of unilateral bronchial stenosis. Continuous pressure leads to retention of secretion, chronic bronchitis and pulmonary fibrosis. The clinical picture may resemble pulmonary tuberculosis. Osler called it aneurysmal phthisis. Pressure on the left recurrent laryngeal nerve often leads to paralysis of the left vocal cord. Hoarseness thus produced may be the first symptom caused by aneurism. Narrowing of the pupil or of the palpebral orifice, and also unilateral sweating may be due to pressure on the sympathetic. Difficulty in swallowing is the result of pressure on the esophagus.

Diagnosis.—Easy as is the recognition of a thoracic aneurism in most

instances, in special cases it may be difficult. The symptoms produced by a mediastinal tumor may be quite similar to those of an aneurism. There may be the same pressure symptoms, and pulsations from the heart

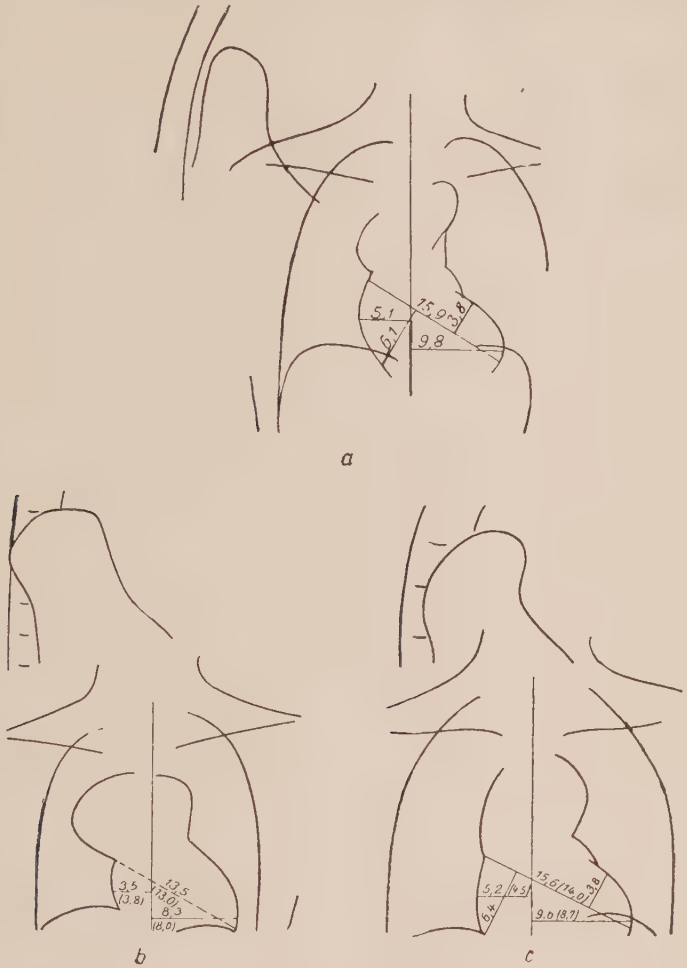


Fig. 129.—Orthodiagrams of aneurisms of the aorta ascendens. The outline of the heart and aorta in the first oblique diameter is shown as well as the outline with dorsoventral illumination. (a) small aneurism recognized only with dorsoventral illumination. (b) large aneurism. (c) middle-sized aneurism recognizable only in the first oblique diameter. The numbers in brackets give the normal average measurements of the cardiac diameters. (Romberg, "Krankheiten des Herzens und der Blutgefässe, Ferdinand Enke.)

or aorta may be transmitted to a tumor. I have seen a malignant tumor destroy the central part of the sternum with the result that a marked pulsation over the front of the chest was produced by the transmission of the cardiac impulse. This lead to the diagnosis of aneurism. A

malignant growth may be pulsatile, but the pulsation is not truly expansile. Furthermore a thorough study of most cases will reveal other signs than pulsation upon which a differential diagnosis can be based.

If on palpation a diastolic shock is felt the diagnosis becomes definite as it is only present in aneurism. (Broadbent.)

Deep-seated aneurism is frequently unrecognized for a long time. Pressure on the bronchi often results in a severe diffuse bronchitis that may be mistaken for tuberculosis or bronchiectasis in the absence of more definite signs of aneurism. Any severe long-continued pain of obscure origin in the chest should suggest the possibility of aneurism and lead to a thorough examination.

The Roentgen examination has proved invaluable in the diagnosis of thoracic aneurism. For details see section devoted to this subject (page 389). The x-ray reveals the existence of many cases with indefinite physical signs. Its employment is essential if the diagnosis cannot be definitely made by other means.

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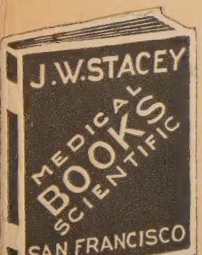
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